RESEARCH TRIANGLE INSTITUTE

PHS Contract No. PH-110-67
Work Order OCD-PS-64-227
OCD Work Unit 3432A

November 1968

FINAL REPORT

R-OU-332

NATIONAL EMERGENCY HEALTH PREPAREDNESS STUDY INCLUDING THE DEVELOPMENT AND TESTING OF A TOTAL EMERGENCY HEALTH CARE SYSTEM MODEL

This document has been approved for public release and sale; its distribution is unlimited.

RESEARCH TRIANGLE PARK, NORTH CAROLINA 27709

RESEARCH TRIANGLE INSTITUTE
OPERATIONS RESEARCH AND ECONOMICS DIVISION
RESEARCH TRIANGLE PARK, NORTH CAROLINA

OCD Review Notice

This report has been reviewed in the Office of Civil Defense and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Office of Civil Defense.

FINAL REPORT R-OU-332

National Emergency Health Preparedness Study Including the Development and Testing of a Total Emergency Health Care System Model

November 1968

Project Personnel

E. L. Hill, A. W. Voors, R. O. Lyday, Jr., J. N. Pyecha,
J. B. Hallan, J. T. Ryan and C. N. Dillard

for

OFFICE OF CIVIL DEFENSE
OFFICE OF THE SECRETARY OF THE ARMY
Washington, D. C. 20310

through

The Public Health Service
U. S. Department of Health, Education, and Welfare

under

Contract No. PH-110-67
OCD Work Unit 3432A

FINAL REPORT

R-OU-332

National Emergency Health Preparedness Study Including the Development and Testing of a Total Emergency Health Care System Model

OCD Work Unit 3432A
Public Health Service Contract No. PH-110-67

RESEARCH TRIANGLE INSTITUTE
Operations Research and Economics Division
Research Triangle Park, North Carolina

Approved by:

Edward L. Hill
Edward L. Hill
Group Leader

Edgar A. Parsons
Edgar A. Parsons, Director

November 1968

ACKNOWLEDGEMENTS

Special appreciation is expressed to:

Dr. Warner L. Wells, Associate Professor of Surgery at the University of North Carolina School of Medicine, for his earlier work in developing the prognosis data on which the present report is based.

Professor Gordon T. Stewart, Chairman of the Department of Medicine and Epidemiology of the Tulane University School of Medicine, Public Health, and Tropical Medicine, for his many helpful suggestions.

Mr. Paul Kaetzel, Project Officer of Public Health Service, for his guidance and assistance, particularly in obtaining and preparing the data used in the New Orleans case study.

ABSTRACT

This study developed a total Emergency Health Care System Model that can be used to study postattack problems in medical preparedness planning for a single locality. The total model consists of two submodels and is capable of analyzing medical system effectiveness, measured by survivors, as a function of medical resources and their employment; e.g., triage, and treatment priorities. The Immediate Effects Submodel analyzes the first 60 days immediately after attack and is applicable to those casualties that survive the initial weapon effects. Casualty types resulting from a specified attack and available medical resources (personnel, facilities, and supplies) serve as input to this submodel. A prognosis based on injury type, availability of appropriate medical personnel, and available medical supplies is applied to these casualties. The number of deaths and survivors, along with the utilization of medical supplies and personnel, are output.

The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats to survivors of the 60 day postattack period throughout the ensuing year. Using a mathematical model of infection, survivors are subjected to the risks of becoming infected by one or more of 16 communicable diseases. A prognosis function based, in part, on the availability of medical staff and supplies required to treat each disease is then applied to the infectives. The model output specifies the number of fatalities and the consumption of medical resources by five-day periods for each disease.

A case study for the total Emergency Health Care System Model was made of the postattack health posture of New Orleans, Louisiana. The hypothetical attack was a surface burst by a 1.5 MT thermonuclear weapon approximately 9 miles south of the center of the city (population of 1,002,000). The results of the one year postattack period for the New Orleans case study indicate that relatively unlimited resources have little effect (few preventable deaths) on the magnitude of deaths among direct effect injured. However, large numbers of epidemic deaths are preventable in the late postattack environment. Since these preventable deaths are highly dependent upon medical resource availability, the importance of preattack medical resource planning and stockpiling of supplies is indicated.

TABLE OF CONTENTS

TABLE OF CONTENTS (Con't)

LIST OF TABLES

LIST OF TABLES (Continued)

LIST OF FIGURES

Chapter 1

Summary

I. INTRODUCTION

A. General

This research, conducted for the Public Health Service (Office of Civil Defense Work Unit 3432A) under Contract No. PH 110-67, was directed toward the development of a total Emergency Health Care System Model that can be used to study and evaluate the nuclear postattack health posture of a single locality. This total model consists of two submodels and can be used in medical preparedness planning for a single locality; i.e., a town, city, or county. It is capable of analyzing medical system effectiveness, in terms of survivors added as a function of the availability and employment (triage and treatment priorities) of medical resources (facilities, personnel and supplies).

The first submodel, th Immediate Effects Submodel, simulates the first 60 days immediately after the attack and is concerned with the handling of casualties that survive the initial weapon effects. Casualties classified by injury type for a specific attack, along with a list of medical resources available in the target area, are put into the submodel, a treatment priority is established, triage is performed, available medical resources are applied, and a prognosis of continued survival or death is derived. Output from the submodel includes the number of deaths and survivors and the utilization rates for medical supplies and personnel.

The second submodel of the Total Emergency Medical Care System Model is the Disease and Chronic Conditions Submodel. This submodel provides a simulated study of the probable generation and effect of communicable diseases among the survivors from 30 days to one year postattack. Using a prognosis function based on a mathematical model of infection and the availability of required medical resources, the model simulates the treatment of infectives and specifies the consumption rate of medical resources, by five-day periods, for each disease.

The Total Emergency Health Care System Model is written in FORTRAN II and occupies approximately 13,000 words of memory. This program was specifically designed to work on the National Civil Defense Computer Facility's CDC 3600, but it can be processed on any computer with a FORTRAN II Compiler and 13,000 words of core storage available. Segmenting of the program into the Immediate Effects Submodel and the Communicable Disease Submodel results in division of the program into two parts of approximately 8,000 and 5,000 words, respectively.

This report consists of 4 chapters and 13 appendices. Chapter 1 contains an introduction to the study and a summary of the study results. The simulation model of emergency medical care in the immediate postattack period is described in

Chapter 2 and the late postattack or communicable disease period in Chapter 3. The results of a case study in which casualty and resource data for the city of New Orleans were processed by the Total Emergency Health Care System Model are presented in Chapter 4.

B. Background

A postattack medical preparedness program must take into consideration the stockpiling of essential medical supplies, establishment of Packaged Disaster Emergency Hospitals, training of professionals and non-professionals in emergency medical care, triage, etc. The number and complexity of variables associated with these various phases of health planning programs indicates the need for developing new and more powerful methods for studying and evaluating medical preparedness.

The initial phase in the development of such methodologies began in 1965 with the initiation of Public Health Service Contract No. PH-86-65-46, Review and Evaluation of the National Health Preparedness Program. Research under that contract examined the nuclear postattack period in terms of a 60-day "immediate nuclear postattack period" and a "late nuclear postattack period" (up to one year). A computer simulation model was developed to study alternative medical strategies in the immediate postattack period. Work related to the late postattack period was primarily directed towards identifying specific diseases most likely to be problematic and towards development of preliminary estimates of the magnitude of the disease problem in the postattack environment. However, because of the preliminary work performed under this initial contract, and the need for further study on the simulation model and the late postattack disease problem, only a summary report from that research was published.[1/] That report, which had limited distribution, did not contain the details of the development of the model input data and other data concerning communicable and chronic disease and emergency medical planning.

1/ Hallan, J. B., J. L. Colley, W. L. Wells, R. S. Titchen, C. N. Dillard, and A. V. Alhadeff. Review and Evaluation of the National Emergency Health Preparedness Program - Final Summary Report, R-OU-209. Research Triangle Park, N. C.: Research Triangle Institute, 30 November 1966.

II. OBJECTIVES AND SCOPE OF WORK

The broad objective of this research was to continue the development of the simulation model capable of studying in depth the cost and effectivenss of alternative strategies for providing medical care and medical support under various postures of nuclear postattack health situations. The model design was aimed at suitability for analyzing medical system effectiveness measured by survivors, for a range of attack conditions, as a function of:

1) medical supplies,
2) medical personnel,
3) medical facilities (including Packaged Disaster Emergency Hospitals), and
4) doctrine of total system employment (triage and treatment priorities).

Detailed objectives of both submodels of the Total Emergency Health Care System Model are described in Chapters 2 and 3; specific tasks associated with developing this model under Public Health Service Contract No. PH-110-67 are:

1) *The casualty simulation phase of the model should extend to at least 60 days postattack including estimation of prognoses data and treatment requirements for radiation injury. Radiation injury is to be considered an initial effect even though dose accumulation may extend to several days or weeks. Since other contemplated studies will consider the added insult of radiation injuries in combination with mechanical trauma and burns, the model should be so constructed as to accept this potential data.*
2) *The disease and chronic conditions phases of the emergency health system should cover <u>all</u> initial effects survivors (injured and uninjured) and carry them forward to one year postattack. This phase includes the selection of the parameters for the 16 diseases selected for further study as outlined in Table XVI of the Final Report prepared under Contract PH 86-65-46 and estimation of upper and lower bounds on the parameters to be studied. Disease propogation models must be programmed for computer solution and disease caseloads must be generated under a variety of assumptions. The model should provide for acceptance at a later date of data relating to possible synergistic interaction between initial injury and later disease.*
3) *The model should be adaptable to the generation of nationally applicable information.*
4) *The measure of system effectiveness (output) should consider only survivors and deaths.*

5) The model should be so constructed as to utilize a full range of resource availability values from minimal to maximum.

6) One (1) case study shall be performed for (1) city as approved by the Project Officer and using Public Health Service supplied data in a mutually agreeable format consistent with the input parameter requirements of the model within one (1) month after request by the Contractor but no later than eight (8) months after effective date of the contract.

7) The model should be adapted to account for complete and full time utilization of medical personnel at those times and places where their efforts would be most effective.

III. MODEL FORMULATION

The approach of the work reported herein was to consider the immediate and late postattack problems capable of being studied with a single Total Emergency Health Care System Model. This model encompasses as submodels the previously developed Immediate Effects Model (with modifications and expansion) and a second model dealing with disease and chronic conditions (developed under the current contract). These submodels cover: (1) the immediate nuclear postattack period; and (2) the time, up to one year after the nuclear weapons attack, during which the surviving population will be faced with problems of diseases and chronic conditions that partly result from the attack and are complicated by a disrupted postattack environment. Figure 1 represents the overall flow and the functional relationships of the Total Emergency Health Care System Model. Details of the approach will be found in Chapters 2 and 3.

In the Immediate Effects Submodel, casualty types resulting from a specified attack are treated by the medical resources (personnel, facilities, and supplies) according to predetermined rules of triage or treatment priorities. The resources may be varied through input data to test the impact of the level of medical stockpiles, Packaged Disaster Hospitals, etc., on the measure of system effectiveness (survivors added by the emergency medical system). Secondary output, available at the option of the user, includes the utilization rate for medical personnel and other specified resources. The simulated community consists of several geographical areas called grids. For the purpose of the model, one of the grids (the hospital grid) contains the total hospital capability of the community; the others contain emergency medical treatment centers. Casualties originate in all grids. A treatment table, consisting of prognosis data, treatment time for injuries, and priorities, is stored in the computer's memory. The table is consulted and available resources applied to casualties (in batches) in order of their preassigned priorities for treatment. Provision is made for the treatment level to be altered depending upon the availability of personnel. The appropriate prognoses are applied to the injured, deaths and survivors are estimated and recorded, and available resources are depleted. The non-hospital grids are processed first, then the hospital grid, and finally, transfers to the hospital grid from the non-hospital grids. Grand totals for the run are prepared and printed out as well.

The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats for a period of about one year postattack. Beginning approximately 30 days postattack, survivors of the Immediate Effects Phase are subjected to the risks of becoming infected by one or more of 16 communicable diseases, using a mathematical model of infection. Remaining medical resources are

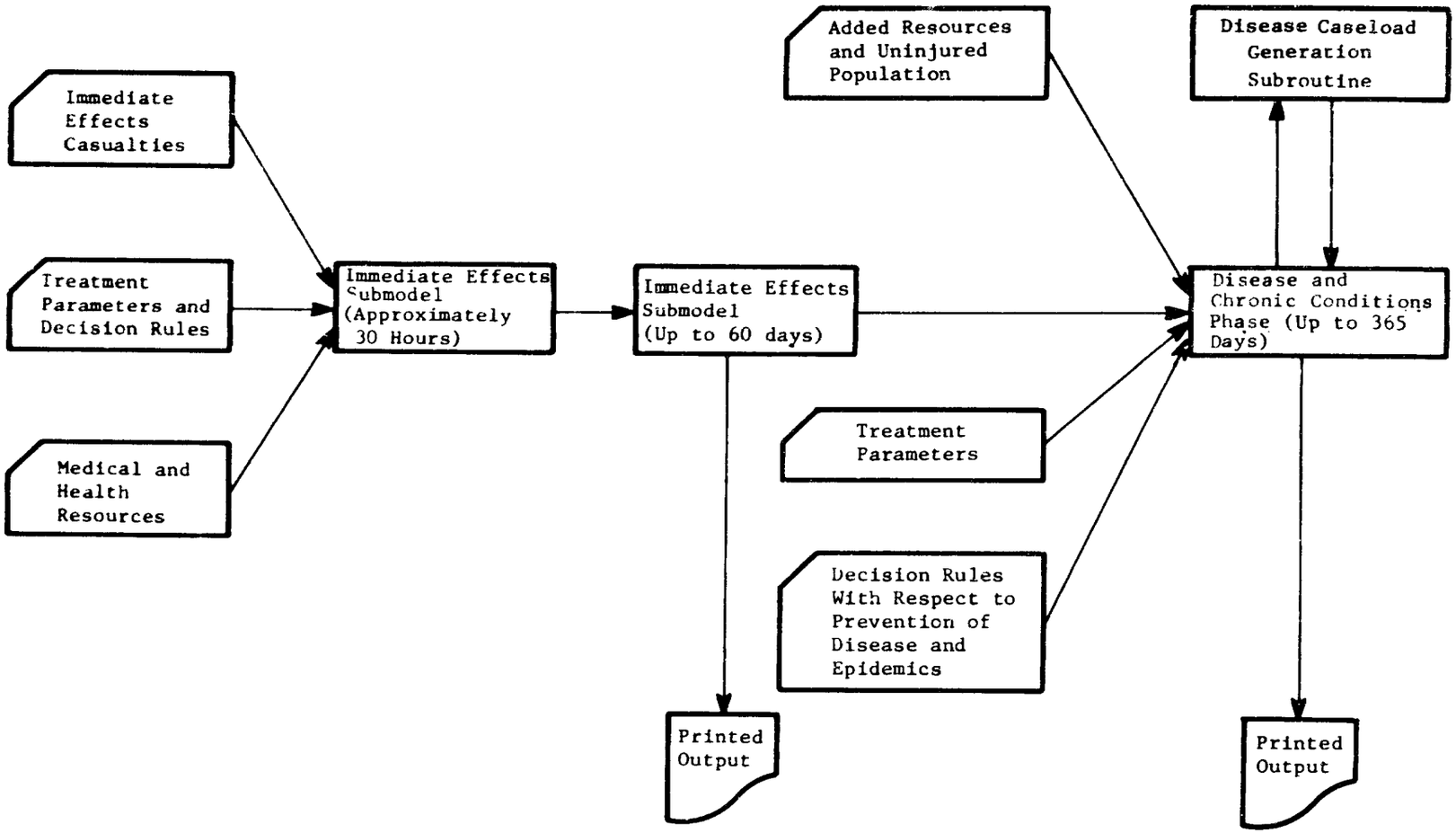


Fig. 1. Overall Flow of Total Emergency Health Care System Model

then applied to the diseased persons, and the health impact created by alternative allocations of the medical stockpiles among the two submodels (Immediate Effects and Disease and Chronic Condition) can be tested by varying the input for these resources. The impact of radiation can be tested by varying the susceptibility to the 16 diseases and the impact of the emergency situation can be tested by varying the intensity of person-to-person contact. A master control card specifies the environmental impact common to the various parameters for each disease. A disease table specifies these parameters for each disease under preattack conditions. The model output specifies the number of fatalities and the consumption of medical resources by 5-day periods.

IV. CASE STUDY

New Orleans, Louisiana (population 1,002,000) was chosen as the test city for the Total Emergency Health Care System Model and the hypothetical attack was a surface burst by a 1.5 MT thermonuclear weapon approximately 9 miles south of the center of the city.

The number and type of injuries expected as a result of the "test case" were obtained by using casualty data from a READY[2/] run and the latest Dikewood injury curves.[3/] Medical supply data were furnished by the Public Health Service and numbers of physicians were supplied by the Office of Civil Defense.

Three simulation runs through the model provided a means of evaluating the postattack health posture of New Orleans (in terms of survivors added) under three assumed levels of medical resources (medical personnel and supplies); i.e., none available, "best" estimates of what would be available, and the required amount for treating all injured at the preferred treatment level (surgeon team level, physician level, allied medical personnel level, or no-treatment level).

Conclusions regarding the findings of the New Orleans Case Study reported in Chapter 4 are as follows:

1) Sophistication of existing medical support systems appears to have little effect in terms of additional survivors during the first 60 days postattack. With the best estimates of the medical resources available in New Orleans, fatalities resulting from the processing by the Immediate Effects Submodel were estimated to be some 48 percent (185,000) of the input caseload (approximately 387,000 people injured by the weapon, either by fallout and/or by direct effects). Processing the model with zero medical resources and total amount for the casualty caseload resulted in 49 percent (188,000) and 47.5 percent (184,000) fatalities, respectively, among the input caseload. Note, however, that better than 90 percent of the fatalities in the New Orleans test case were due directly or indirectly to fallout and medical care did not affect the subsequent deaths. Thus, the conclusion of this case study was that the medical system was not a factor since an expanded medical system would be able to decrease deaths among the initially surviving injured by only 2 or 3 percent.

2/ National Resource Analysis Center, Office of Emergency Planning, READY 1: Summary Analysis, Category H&C, New Orleans SLA With Terrain Shielding (unclassified). Washington, D. C.: Executive Office of the President.

3/ Davis, L. Wayne, et al. Prediction of Urban Casualties and the Medical Load From a High-Yield Nuclear Burst. Albuquerque, N. Mex.: The Dikewood Corporation, December 1967.

2) Medical resources become far more critical in the late postattack period for the nine likely disease threats in the New Orleans area. The results of the Disease and Chronic Conditions Submodel, using maximum, available, and minimum medical resources, indicate that deaths from disease among the population surviving the first 60 days postattack were 2 percent, 4 percent, and 35 percent, respectively.

3) The critical nature of the medical resource problem implies that careful deployment and prudent conservation are needed in the postattack period if the devastating impact of disease threats is to be minimized.

V. CONCLUSIONS AND RECOMMENDATIONS

A. Conclusions

An operational two phase simulation model of the nuclear postattack total emergency health care system capable of examining the postattack period out to one year has been developed and tested. The Total Emergency Health Care System Model provides a flexible tool for examining in detail the health and medical care problems likely to exist in the immediate postattack period due to the direct effects casualties (Phase 1) and in the late postattack period because of disease and chronic conditions (Phase 2). The model is currently capable of studying problems in areas as large as a metropolitan area.

Injured casualties serve as input to Phase 1; Phase 1 survivors and the uninjured population are input to Phase 2. In both instances a range of medical caseloads (injuries, radiation casualties and disease victims) and resources (personnel, supplies and facilities) can be used in the model. Model output allows examination in detail of personnel and medical supply utilization. Postulated alternative systems can be compared in terms of survivors added by various strategies.

B. Recommendations

Based upon the findings of the current study, it is recommended that further research in this area be continued to (1) improve the existing Total Emergency Health Care System Model, and (2) to develop an appropriate model for study of large geographic areas.

1. Improvements to the Existing Emergency Health Care System Model

a. Input Data Generators

The existing Emergency Health Care System Model is hampered in its application because of the time and effort required to prepare the necessary detailed casualty and medical resource input data. This problem can be overcome by the development of input data generators for casualties and medical resources. Input data generators are in effect computer programs which facilitate preparation of complex data for ultimate use in a separate model.

Currently, casualty input data for the model must be hand generated from existing casualty assessment programs to provide detailed injury estimates by geographic location. This operation is tedious and hampers application of the model. It appears feasible to develop a computer program to prepare output data from existing damage assessment programs for use as direct inputs to the Total Emergency Health Care System Model.

The existing model also requires detailed information concerning the existence and location of medical resources including facilities, personnel and supplies; presently there is no single source which can provide such data. It does appear, however, that by accumulating certain basic medical data concerning a given metropolitan area, the resources of that area and the damage from a range of nuclear attacks to those resources may be synthesized by a computer program into a form usable by the model.

Accordingly, it is recommended that future work include the development of casualty and medical resource input generators to facilitate further study and application of the model.

b. Measures of Effectiveness

There are many practical and conceptual problems surrounding the use of measures of effectiveness in computer simulation models. While such measures are obviously needed to measure system response to various situations being depicted by the model, they are usually incapable of fully describing the response. The effectiveness measure of the current model is that of simple survivors and fatalities that are produced by the system. It is a gross but nonetheless effective measure of the ability of the system to handle complex interaction of demands and resources.

It follows that having once determined the number of survivors which may result from a given system, consideration should be given to the quality of survivorship. It is, therefore, recommended that appropriate measures be examined which are capable of describing the survivor in terms of productivity, efficiency, and disability in a postattack period.

2. Development of an Aggregate Health Care System Model

The current Emergency Health Care System Model is designed for a single city application and is not necessarily directly applicable to studies of larger geographic areas, such as those of a state, regional or national scale. This is due primarily to the necessary assumption that all hospital capabilities are located in one "grid;" i.e., the smallest geographic area in the simulation.

For example, if a state were analyzed, one Standard Location, city, or county would be assumed to contain all hospital facilities. The validity of this assumption relative to transportation of casualties to hospital facilities is questionable when an area larger than a city is simulated.

The methodology and implications of the current model may not be appropriate for studies of a state, regional or national basis even if the current model was "scaled up" to evaluate such larger geographic areas. Therefore, it is recommended that a complete and independent analysis be made within the existing state-of-the-art of the possibilities of modeling the postattack health and medical problems for geographic areas larger than a single city. Such a study should include development of parameters important to national survival and individual implications of a city by city analysis integrated into a study of larger geographic areas.

Chapter 2

Immediate Effects Submodel

I. INTRODUCTION

In the earlier study, a model was programmed and debugged which can be used to determine the effectiveness of alternative medical support systems in the first 30 hours of the postattack period. This model did not account for the continuing load placed on the system by medical care following the initial treatment. The Immediate Effects Submodel developed under the current contract was improved and is capable of describing all essential elements of a medical support system out to a period of 60 days postattack. It is capable of examining the final prognosis of the direct effects and radiation casualties using varying levels of medical resources; i.e., maximum, estimated available, and zero numbers of medical personnel and supplies.

The submodel simulation program operates in the following manner. A community is considered to consist of several geographical areas called grids. One of the grids is the hospital grid; i.e., it contains the total hospital capability of the community. A treatment table, consisting of prognosis data and treatment times for the injuries that are to be considered in the simulation run, is input into the computer's memory. Available resources for the particular area under study are applied to the casualties in the order of their preassigned priorities for treatment. Provision is made for the treatment level to be altered depending upon the availability of personnel. Based on the level of available medical resources, the appropriate prognoses are applied to the injured, estimated deaths and survivors are recorded, and the available resources are allocated. Each grid is processed using resources within the grid and then transfers of selected casualties are made to the hospital grid from the non-hospital grids for processing. Casualties surviving at the end of 60 days, as well as non-injured survivors, are input for the Disease and Chronic Conditions Submodel.

II. SUBMODEL INPUT AND OUTPUT DATA

A. General

The Immediate Effects Submodel describes all essential elements of a medical support system in a single locality, such as a city or county. In this simulation, the measure of effectiveness of the medical support system was taken to be survivors added by the medical system. The inputs are the casualty types and numbers resulting from a specified attack and various levels of medical resources (personnel, facilities, and supplies); the output is survivors. Another practical output, at the option of the user, may include utilization of medical personnel, and other specified resources[1/] (e.g., hospital facilities and pharmaceutical supplies).

Figure 2, Basic Inputs and Outputs of the Immediate Effects Submodel of the Total Emergency Health Care System Model, summarizes the planner inputs needed and submodel outputs which are provided. These are discussed in detail in the following sections.

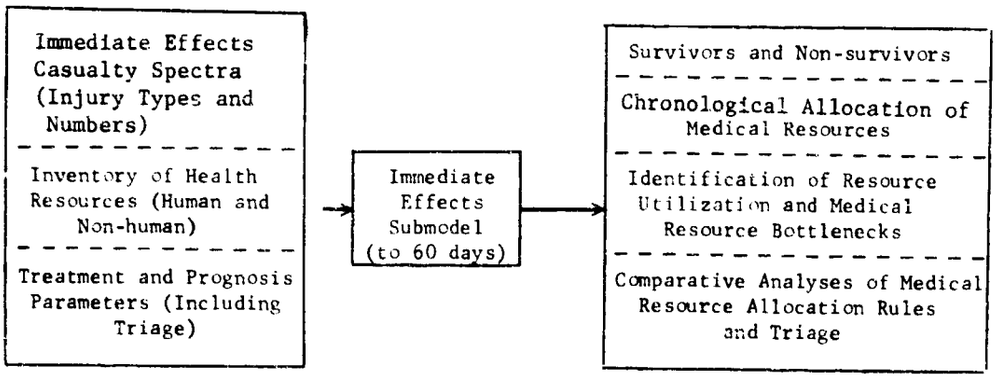


Fig. 2. Basic Inputs and Outputs of the Immediate Effects Submodel.

1/ A glossary of terms used to describe the essential items and events of this submodel will be found in Appendix A.

B. Input Data

1. Casualty Load

The injury caseload is prepared manually for input to the model. Each injury is defined in sufficient detail to allow specification of care requirements and prognoses under a variety of treatment options, as described further below.

It was originally anticipated that detailed information would be available for input data from ongoing studies concerning specific types of injuries as a function of shielding and weapon parameters. However, these studies were not sufficiently advanced to provide the required data during the course of this study.

For case studies with the model, injury data are developed using a postulated burn and mechanical injury spectrum, Dikewood casualty curves,[2/, 3/] and assumptions regarding resident construction and population. Protection data (shielding posture) and the population data base are developed from conventional sources (National Fallout Shelter Survey, U. S. Bureau of the Census Data, the OCD Five-City Study, Community Shelter Plans, etc.). An example of injury data preparation for processing by the model is contained in Chapter 4 and Appendix D.

2. Medical Resources

Estimates of surviving medical personnel and supplies are also prepared manually by the user through the application of damage assessment techniques to preattack personnel and supplies. An example of this is also contained in Chapter 4.

3. Prognoses

A medical prognosis is required for each injury. The prognosis is an estimate of the probability of death from an injury as a function of both the kind of treatment employed and the time delay before treatment. Prognosis data estimates for the injuries used in the model are presented in Appendix B. Numerical values were estimated by Dr. Warner Wells of the University of North Carolina School of Medicine.

2/ Davis, W., et al. Development of Typical Urban Areas and Associated Casualty Curves. Albuquerque, N. Mex.: The Dikewood Corporation, 1965.

3/ Davis, W., et al. Prediction of Urban Casualties [illegible]mediate Effects of a Nuclear Attack. Albuquerque, N. Mex.: The Dikewood [illegible]oration, 1963.

The prognosis for each injury as a function of time takes the general form illustrated in Figure 3.

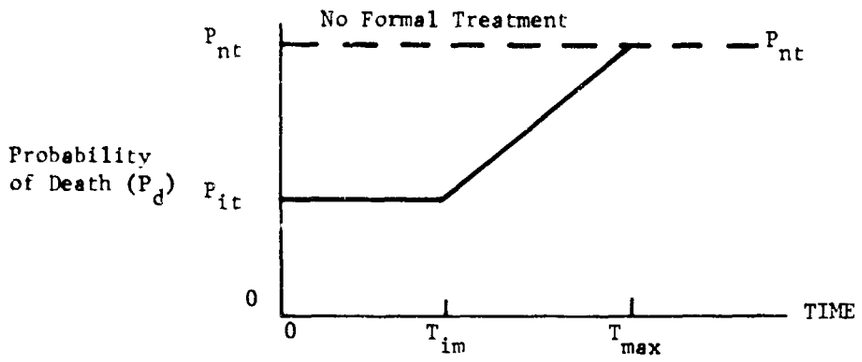


Fig. 3. Injury Prognosis

As can be seen, the prognosis function is composed of three segments: (1) time zero to T_{im}, during which a specified treatment is maximally effective and probability of mortality is lowest; (2) T_{im} to T_{max}, during which the prognosis steadily decreases; and (3) beyond T_{max} when the probability of death with treatment equals that with no treatment (typically $T_{max} = 2 \times T_{im}$).

4. Preferred and Actual Level of Treatment

Several "levels" of treatment are available in the model. These include surgical teams, physicians, allied medical personnel, or no-treatment.

One of these defined levels of treatment is specified as the "preferred" treatment for each injury type (see Appendix B). The preferred treatment is the lowest level which will not greatly reduce the chances for survival of the injured person.

Availability of personnel and supplies affects the actual treatment provided to the injured in the model. Thus, it was necessary to establish as input a table of the decision rules and alternative prognoses for various treatment options for each of the injury types considered. The decision rules specify for each injury type (1) the preferred treatment and (2) alternatives to be used when the preferred treatment is not available.

As demands for treatment exceed the number of medical personnel available and waiting lines form, the time which a casualty must wait for treatment increases. The program calculates the waiting times; if it exceeds T_{im} (that initial period during which a delay in treatment does not alter survival probabilities), then the prognosis under conditions of delayed treatment is calculated. This prognosis is compared with the prognosis calculated using the level of care downgraded to the next lower level. If the probability of death is less using the lower level of care--which may be the case if the lower level of care will be available sooner--the casualty is assigned to the next lower level of care.

The procedure is illustrated in Fig. 4. Assume the calculated time-to-treatment under the preferred level (physician care) is T_P, with corresponding prognosis P_P. Since $T_P > T_{im}$, prognosis under the downgraded level (allied medical personnel) is then determined using T_A (time-to-treatment by allied medical personnel); if $P_A < P_P$, then treatment is provided by allied medical personnel outside the hospital. If this downgraded level is also saturated to a point where the prognosis cannot be improved by downgrading, the next higher quality of treatment level is examined. If all alternate levels of care are saturated to a time greater than T_{max}, the injured are transferred to the hospital.

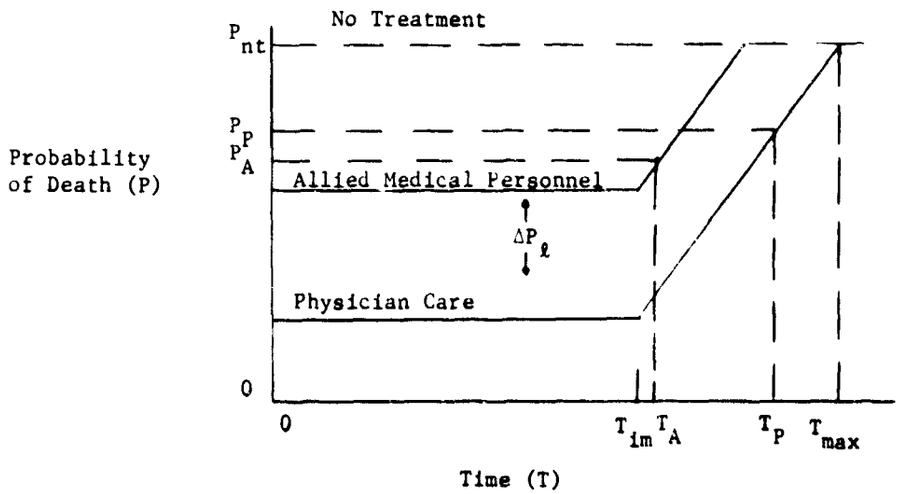


Fig. 4. Injury Prognosis for Two Levels of Treatment (Physician Care is the Preferred Treatment in this Example).

In the hospital, injured are treated at the preferred level of treatment unless that level is saturated. In these cases, alternatives are examined as above.

5. Priority Rule

An essential aspect of disaster medical treatment is triage[4/], the sorting of the injured into categories according to treatment priorities. Effective triage will increase the number of survivors subject to the constraints of limited medical resources.

The approach to triage in this study involved consideration of ΔP_{ℓ} (the degradation in probability of survival owing to downgraded care), T_T (time required for treatment) and T_{im} (the time period during which delay of treatment does not degrade the prognosis). A highest priority patient would be one with minimal T_{im} and T_T, and maximum ΔP_{ℓ}. The priority function, which ranks injury categories in order of priority for treatment within each preferred treatment level, is:

$$\text{Treatment Priority} = \frac{\Delta P_{\ell}}{T_{im} + T_T} \text{ ; high values mean high priority for treatment.} \qquad (1)$$

Priority values associated with each injury will be found in Appendix B.

C. Output Data

The submodel output contains the following information for each grid (including the hospital grid) and for the total community.

1) The number of injured (including fallout) by preferred treatment levels.
2) Treatment actually furnished to the injured; e.g., level of medical treatment with or without supplies, downgraded treatment, upgraded treatment, no treatment, and sent to hospitals.
3) Disposition of casualties (survivors or fatalities).
4) Medical treatment packages initially available, used, and remaining.
5) Medical personnel hours available and the hours used for each treatment level.

Details concerning the precise use of input cards and examples of output format for the Immediate Effects Submodel are presented in Appendix D and in Chapter 4.

4/ "Development and Recommendation of Criteria Needed as a Basis for a National Emergency Medical Care Plan Under Terms of Contract No. CD-SR-58-1." Annex H. Chicago: American Medical Association, 1957.

III. SUBMODEL ORGANIZATION AND LOGIC

A. General

This section describes the basic information flow through the Immediate Effects Submodel and the logic with which the model uses this information to simulate the treatment of immediate effects casualties. Certain aspects of the simulation logic are complex enough to warrant special emphasis; these include triage, "downgrading", and the "priority" logic for follow-on treatment. The overall logic is described in sufficient detail to provide the reader with a working knowledge of all of the major decisions and logical loops used in the Immediate Effects Submodel.

B. Functions of the Submodel

The Immediate Effects Submodel is designed to perform the following functions:

1) Treat the casualties resulting from a nuclear attack, according to treatment priority rules and available level of treatment (preferred and actual) and by subdivision of the district under study (grid).
2) Record the consumption of medical treatment packages and of personnel time in the treatment of casualties, by subdivision of the district under study (grid).
3) Determine and record the numbers of deaths resulting at all levels of treatment (preferred and actual) according to the stated rules and probabilities, subject to the availability of supplies and personnel in the initial treatment phase and the follow-on phase.
4) Indicate the number of casualties for which supplies of medical supply packages are not available.
5) After storing the death figures of a base run, indicate for each subsequent run the number of survivors added by changes in the disposition of personnel and supplies.
6) Identify bottlenecks in medical personnel, drugs, and supplies which cause cessation of required follow-on treatment to casualties.

C. Submodel Logic

1. Data Flow

A highly simplified version of the logic for the simulation model is shown in Figure 5 and is described below (see Appendix C for a detailed flow chart of the Immediate Effects Submodel).

Step 1. The simulation run is initiated by reading in a Master Control Card and a Treatment Table. The parameters on the Master Control Card indicate the duration of Phase I initial treatment (in hours) and the duration of

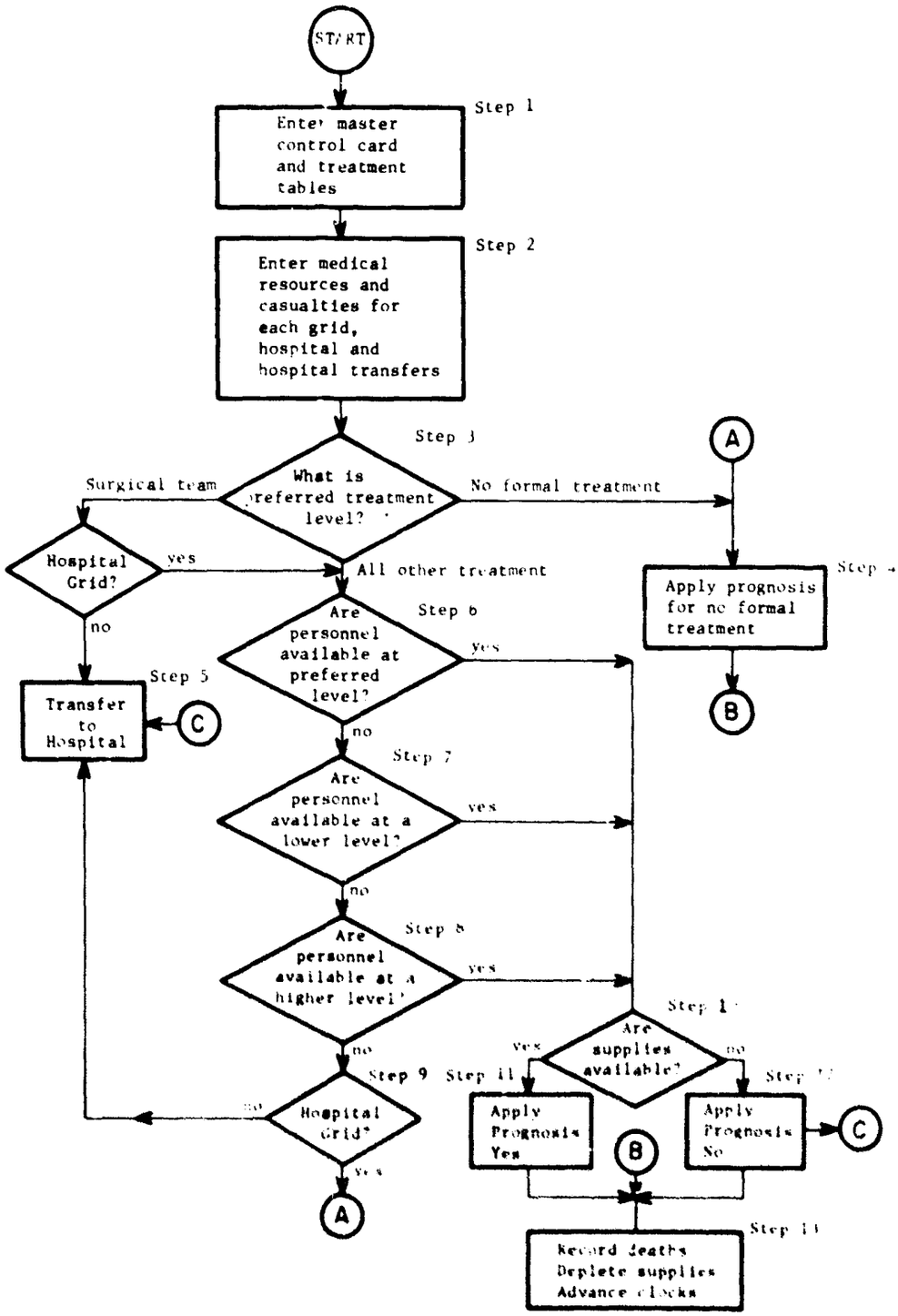


Fig. 5. Immediate Effects Submodel Flow Chart

Phase II initial treatment (in days). The first phase of the initial treatment begins at the time the first casualties report for treatment and continues throughout the 30 hour period during which treatment personnel are available on duty continually and without rest. During Phase II of the initial treatment (from 30 hours up to 60 days postattack), medical personnel are assigned on a half-time basis (i.e., 12 hours per day).

The Treatment Table contains a description of each injury and the rules regarding the disposition of casualties with that injury (Appendix B). This descriptive data includes:

1) The injury code number.
2) The time in hours needed to treat a casualty suffering from this injury.
3) The time period in hours following which treatment no longer will be effective.
4) The initial period in hours during which delay in providing treatment does not increase the probability of death.
5) The code number (1 through 16) of the medical treatment package prescribed for this injury.
6) The level of treatment (1 through 4) proper for this injury.
7) The differential by which the probability of death is increased by downgrading treatment to the next lower level of personnel.
8) The differential by which the probability of death is increased by travel to the hospital through fallout, etc.[5/]
9) The probability of death if treatment is given immediately.
10) The probability of death if treatment is not given.

The length of the time by which earlier treatment at the next lower level would result in a lower probability of death is calculated for each injury code number at the same time that the Treatment Table is read into storage.

Step 2. Resource and Casualty data cards are read into the simulation program. These data describe each batch of casualties reporting to an Emergency Treatment Center and indicate the resources (personnel and supplies) available for treating these casualties.

Step 3. For every grid the model first considers the injury category with the highest priority, then the next highest, etc., and applies the indicated level of preferred treatment.

Step 4. If no-treatment is the preferred level of treatment (either because the injury is minor or very severe), the proper prognosis is applied and

---

5/ This option, although available in the model, was not used because of the present level of knowledge concerning the noxious effects of transportation under these circumstances.

entries are made in the appropriate records.

Step 5. If a surgical team is the preferred level of treatment, the cases located in a nonhospital grid are immediately transferred to the hospital since surgical teams are only available in the hospital grid. This model assumes that there are no travel restrictions between nonhospital grids and the hospital; e.g., vehicles and fuel are available, roads are negotiable, etc.

Step 6. If the case is to be treated within the grid (hospital or nonhospital), the model first determines whether or not personnel are available at the preferred level.

Step 7. If personnel are not available at the preferred level, a check is made for persons at the next lower treatment level.

Step 8. If personnel are not available at the lower level, a check is made for the availability of higher level personnel.

Step 9. If all three levels are not available and the patient is not already in a hospital, the case is transferred to the hospital. If all three levels are not available in a hospital grid, no formal treatment is applied. A lower level of care than the preferred level degrades the prognosis; a higher level leaves the prognosis unchanged.

Step 10. After the level at which treatment will be administered is determined, a check is made for the availability of supplies.

Step 11. If personnel and appropriate supplies are available, the prognosis is applied.

Step 12. If treatment is to be given without prescribed supplies, the prognosis associated with the treatment level is reduced to a point midway between the prognosis with supplies and that with no treatment. An option available in the step provides that if the required medical supplies are exhausted, designated casualty categories are transferred to the hospital.

Step 13. After applying the prognosis as indicated in either Step 11 or Step 12 above, deaths are recorded, supplies are depleted, and personnel time clocks are advanced to record the times spent in treatment.

After all nonhospital grids have been processed through the model, the hospital transfers are analyzed by separate runs through the same program logic.

The model is further designed to store the fatality figures resulting from the first simulation run so that the difference between these and the numbers of fatalities produced by alternative emergency medical systems can be calculated and printed as the "NUMBER OF ADDED SURVIVORS".

2. Decision Rules

Decision rules built into the model include:

1) Casualties for whom the preferred level of treatment is "no formal treatment" consume neither supplies nor personnel time; therefore, the probability of death without treatment is applied to them. No formal treatment has been designated as Treatment Level 1 in the model.

2) Casualties arising in a nonhospital grid and requiring surgical team treatment (Treatment Level 2) are transferred to the hospital grid. As previously discussed, this transfer process increases their probability of death. When the waiting time required for surgical team treatment in the hospital exceeds the T_{max} time period for the casualties, the probability of death with the added differential for downgraded treatment by physicians is considered. If the probability of death associated with immediate treatment by physicians is lower than that associated with delayed surgical team treatment, the preferred treatment level is downgraded from Level 2 (surgical teams) to Level 3 (physicians). There is no provision for seeking a lower probability of death by further downgrading to Level 4 (allied medical personnel).

3) Treatment level 3 casualties are treated by physicians unless downgraded treatment by allied medical personnel is available at a lower probability of death because it can be applied sooner.

4) Treatment Level 4 casualties are attended by allied medical personnel and downgrading is not possible.

5) For Treatment Levels 2, 3 and 4, the first step is to check the inventory of medical treatment packages in the Emergency Treatment Center or hospital. The inventory is reduced by the quantity which will be consumed in treating the batch (or part of the batch). The casualties for whom medical treatment packages are not available, if not transferred to the hospital (a decision entered in the Treatment Table), are segregated to be attended by appropriate medical personnel but with an increased possibility of death. A record is made of the number of casualties lacking supplies which indicates whether or not they were transferred to the hospital for this reason.

6) In cases where lack of personnel at a treatment level (other than surgical team) precludes the possibility of treatment at that level, the possibility of finding personnel available at a higher level is

investigated. This upgrading is limited to the next higher level and does not change the probability of death.

7) At all treatment levels in nonhospital grids, when it is determined that treatment will not be given because personnel are not available or downgrading is not feasible, the batch or part of the batch is transferred to the hospital. Similarly, in the case of the hospital, the casualties are consigned to the untreated category and the probability of death without treatment is applied in the absence of treatment personnel. In both cases, whatever supplies were removed from inventory in anticipation of treating these casualties are returned.

8) All casualties whose treatment can be started within the time period $T_o - T_{im}$ have applied to their group the probability of death with immediate treatment.

9) Casualties whose treatment can be started only after the time period T_{max} have applied to their group the probability of death without treatment.

10) Casualties whose treatment is started during the period $T_{im} - T_{max}$ receive the average probability of death (the probability at the midpoint of the time span required to treat the entire batch).

11) Casualties who receive attention from medical personnel but without the proper medical treatment packages have the probability of death increased to the point halfway between that probability already determined by the rules stated above and the probability of death without treatment.

12) In every case where a differential is added to a probability of death, the probability of death without treatment is the maximum to which it may be increased.

Chapter 3

Disease and Chronic Conditions Submodel

I. INTRODUCTION

This Chapter is concerned with the Disease and Chronic Conditions Submodel of the Total Emergency Health Care System Model. This submodel is designed to model the generation and effects of likely disease threats for a period of about one year postattack.

The health problems considered in this Chapter are those that begin several days after the attack--when the acute medical problems associated with the immediate effects of the attack are subsiding--and end a year after the attack. In order to determine precautionary measures that would best promote health in this period, attention was limited to diseases which can cause death in that time. Preventable deaths were thus used as a single criterion in predicting the effects of any such measures.

Two classes of disease were considered with regard to health problems arising during this postattack phase; i.e., communicable diseases and noncommunicable or chronic diseases.

This Chapter touches only briefly on disease threats during the immediate effects period, as this has been previously studied. Life-threatening health problems in the Immediate Effects Phases will be relatively insignificant.[1/] There will be little opportunity for disease to be introduced and insufficient time for most life-threatening diseases to produce secondary infections (other than high prevalence of upper respiratory infections). Life threats will largely apply to infants, the elderly, and the infirm during this phase. Thus, the estimations of loss of life due to postattack disease and chronic conditions were limited to the period beginning 30 days after the attack.

Insight into the following kinds of questions can be provided by this submodel.

1) What is the sensitivity of the number of survivors/nonsurvivors to varying strategies of the distribution of stock piled medical supplies between the Immediate Effects Period and Disease and Chronic Conditions?
2) What is the sensitivity of the number of survivors/nonsurvivors to varying strategies of strict quarantine enforcement resulting in the elimination

---

1/ Herzog, W. T. Emergency Health Problems Study, Final Report R-OU-106. Durham, North Carolina: Research Triangle Institute, July 31, 1963.

of the more esoteric diseases; e.g., smallpox and plague?

3) What is the effect of increasing the numbers of medical personnel and/or medical supplies?

4) To what extent do various levels of fallout radiation and decontamination affect the number of survivors?

5) What are the bottlenecks in epidemic prevention?

II. BACKGROUND

A. Introduction

The background material which follows in this section is largely a review of the relevant measures and outcomes developed in the previous contract (PH-86-65)[2/], which formed an integral base for the modeling efforts described in Section V.

A first approximation of the scope and magnitude of the communicable disease problem was obtained by the following two-step process:

1) Elimination of all infectious diseases unlikely to contribute markedly to death during this period. This reduced the number of relevant diseases to sixteen.

2) An exhaustive literature survey that led to the estimation of three factors for each of the sixteen diseases: (a) the probability that there is a focus of the infectious organisms in a reference population of 20,000; (b) the fraction of this population expected to contract the disease under the specified conditions; and (c) the case fatality rate under these conditions. Fatalities were estimated by multiplication of these three factors.

A similar procedure was also used in studying noncommunicable or chronic diseases: (1) limiting the study to the ten disease classes expected to contribute most to postattack fatalities, and (2) estimating fatalities by multiplying expected postattack prevalence with preventable case fatality rates.

The resulting fatality figures show that the estimated preventable death rate from non-communicable-and-chronic diseases is certainly small as compared with the communicable disease threat.

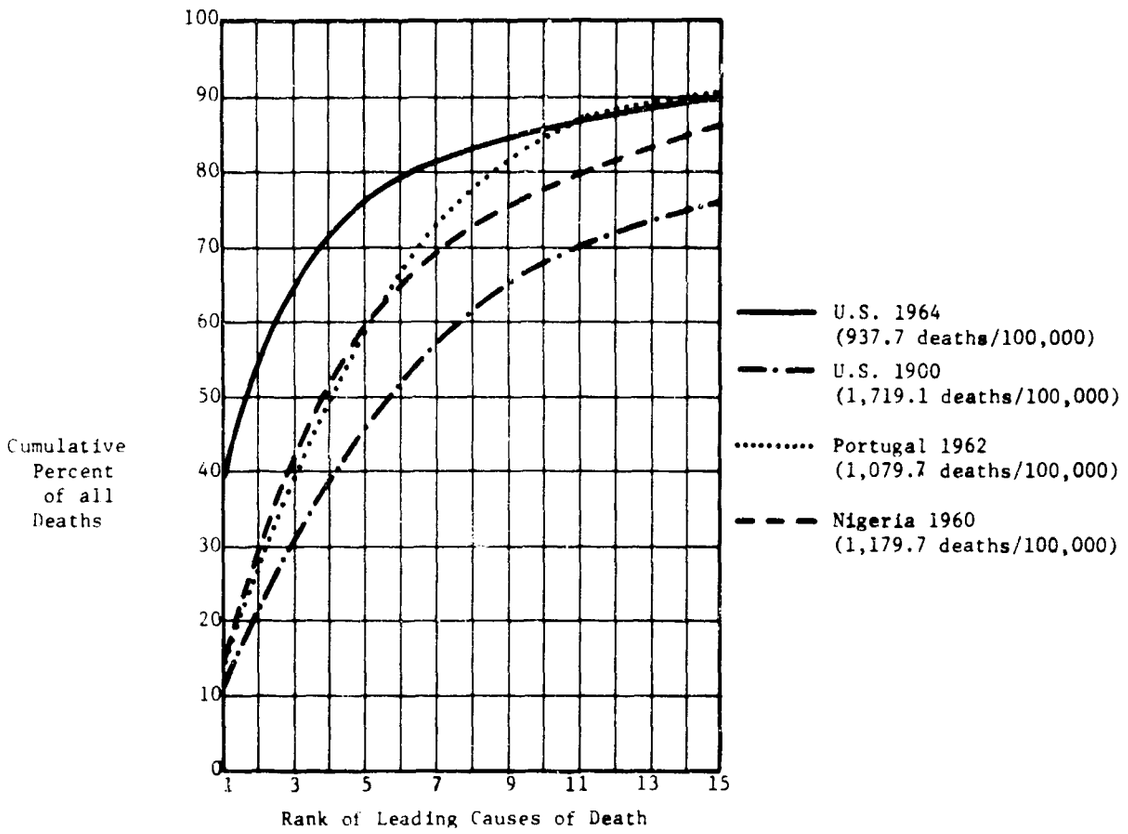
B. Approach

Although the goals of a normal peacetime health system are to provide total well being, they are more modest in an austere postattack environment. The initial measure of effectiveness chosen was survival. Further, no distinction was made among survivors--young, old, male, female, etc. Each type contributes one unit to the measure of effectiveness chosen. It was concluded that later improvements might recognize man-days lost due to illness, permanent disability, etc.

2/ Hallan, J. B., J. L. Colley, W. L. Wells, R. S. Titchen, C. N. Dillard, and A. V. Alhadeff. Review and Evaluation of the National Emergency Health Preparedness Program - Final Summary Report, R-OU-209. Research Triangle Institute, Research Triangle Park, N. C., 30 November 1966.

The problem was severely constrained initially in order to obtain rapidly some approximate results which might later be refined. This initial simplification was as follows:

1) Attention was limited to diseases which can cause death in the first year. This ruled out veneral diseases and tuberculosis (new cases), since only in rare cases (among the very old and the very young) does death ensue in less than one year. This simplification also excluded deficiency diseases (scurvy is the only one which has a significantly high fatality rate). Starvation and death due to thirst were ignored as they are improbable except in isolated instances.
2) The effects of radiation (alone or combined with disease) were ignored initially because the synergistic effects of radiation and disease are much less well known than either alone.
3) The hypothesis was made that a small number of diseases (communicable alone, non-communicable alone, or both) account for the majority of the postattack threat to life. The screening method used to identify this small number is described later. This procedure concentrated the efforts of the project's medical consultants on these few diseases (16 communicable and less than a dozen non-communicable and chronic; see Tables I and II on Pages 3-8 and 3-9, respectively). Support for this hypothesis can be found in Figure 6, which compares the frequencies of causes of death in countries with variable health environments. The number of causes required to reach any percentage of the total deaths is greater under more primitive conditions. However, even in the U. S. in 1900, six causes account for 50 percent of the total. This listing of the diseases in Figure 6 is found in Appendix E.



Sources: Sartwell, Philip E., ed. Maxy-Rosenau: Preventive Medicine and Public Health, 9th Edition. New York: Appleton-Century-Crofts, 1965, p. 507.

National Office of Vital Statistics, Vital Statistics of the United States, 1964. Washington, D. C.: U.S. Government Printing Office, 1966, Vol. 1, Section 1, p. 16.

Statistical Office of the United Nations, Demographic Yearbook, 1963. New York: United Nations, 1964, pp. 592 and 609.

Fig. 6. Cumulative Frequency Distribution of Total Deaths by Fifteen Leading Causes of Death in the United States, Nigeria, and Portugal. (Note: See Appendix E for a listing of the specific fifteen causes of death for each country.)

C. Communicable Disease

1. General Background

Although the great causes of death in the United States today are no longer communicable disease, history furnishes ample evidence that these can be significant threats to health and to life under primitive conditions and therefore, must be considered a potential threat in the disrupted postattack world.[3/, 4/, 5/]

It appears that mathematical modeling of disease spread is not possible *a priori*; rather the models may be (and have been) used to fit an observed epidemic, and thence to estimate the parameters characterizing the disease. Theory does, however, provide a framework in which to think systematically of the problem. The following three basic modes of disease spread have been observed by epidemiologists over the years; and may be considered as derivatives from the mathematical theory of epidemics:

1) Man-to-man (e.g., influenza).

2) Food- or water-to-man (e.g., botulism).

3) Vector-to-man (e.g., plague via rat fleas).

There is a fourth class that is considered to be of far less importance in this study; i.e., the organism has a significant lifetime outside a living host, and, hence, may be thought of as being present in the environment (tetanus). However, in considering all four of these classes, it must be emphasized that some diseases have more than one mode of transmission.

The number of fatalities resulting from these diseases can be estimated by the following formula:

$$\left[\begin{array}{l}\text{Probability of a source of the infectious organism in the local environment}\end{array}\right] \times \left[\begin{array}{l}\text{Expected number of the group contracting the disease under the specified conditions}\end{array}\right] \times \left[\begin{array}{l}\text{Case fatality rate under the specified conditions}\end{array}\right] = \left[\begin{array}{l}\text{Expected number of fatalities}\end{array}\right] \quad (1)$$

3/ Ackerknecht, Erwin H. History and Geography of the Most Important Diseases. New York: Hafner Publishing Company, 1965.

4/ Mitchell, H. H. Survey of the Infectious Disease Problem as it Relates to the Postattack Environment, RM-5090-TAB. Santa Monica, Calif.: The RAND Corporation, August 1966.

5/ Mitchell, H. H. Plague in the United States: An Assessment of Its Significance as a Problem following a Thermonuclear War, RM-4968-TAB. Santa Monica, Calif.: The RAND Corporation, June 1966.

This concept, by itself, is useful in structuring the problem by identifying the impact of a particular preventive measure or treatment. Additional principles are known which aid ir estimating the threat. These details are discussed in Appendix F.

2. Results of Initial Screening

An initial four-step screening procedure was conducted to obtain a small list of potentially critical diseases for more detailed analysis. The factors considered were those discussed previously, and summarized by Equation (1). From a list of 126 communicable diseases or disease groups cited in Gordon[6/], diseases were eliminated as follows:

1) Those which are not endemic to the United States; e.g., loiasis and yaws. This was equivalent to eliminating those diseases with a negligible probability of an initial case. Of the 126 considered, 36 were eliminated on this basis.
2) Those which have a low historical incidence; e.g., dracontiasis and histoplasmosis. This was equivalent to eliminating those which have either a low probability of an initial case in a community, or which are negligibly infectious (small expected size of epidemic), or both. This procedure screened out 40 diseases. (This assumed current immunization status for polio.)
3) Those which have a low untreated case fatality; e.g., herpangina and hookworm disease. The cutoff value was taken to be a 2 percent untreated case fatality rate, except for diseases having a very high historical incidence (only influenza and hepatitis) where the fatality rate cutoff was taken to be about 1 percent. This criterion eliminated 43 diseases.
4) Those of a final group that require many cases to initiate an epidemic (3 rejected on this basis) or are very difficult to transmit (10 rejections).

This left a list of 16 diseases for further analysis; these are identified in Table I. Estimation of postattack disease parameters, effects of counter measures and preventable deaths are discussed in Appendix G.

6/ Gordon, John E. Control of Communicable Diseases in Man. New York: The American Public Health Association, 1965.

Table I

CHARACTERISTICS OF A SELECTED LIST OF SIXTEEN POTENTIALLY CRITICAL COMMUNICABLE DISEASES

Disease	Brief Description	Mode of Transmission	Immunization	Treatment
Pneumonoccal Pneumonia	An acute bacterial disease of the lungs characterized by sudden onset of chill followed by fever	man-to-man	None	Antibiotics
Influenza	An acute infectious disease of the respiratory tract characterized by abrupt onset of fever, chills, headache, muscle pain, and sometimes prostration	man-to-man	Partial (Vaccine)	No specific treatment
Typhoid	A generalized bacterial infection characterized by malaise, slow pulse and enlargement of spleen, loss of appetite, weakness, sometime diarrhea, always malabsorption of nutrients	enteric man-to-man	Vaccine	Antibiotics
Paratyphoid B	A generalized bacterial disease characterized by continued fever, enlargement of spleen, and usually diarrhea	enteric man-to-man	Vaccine	Antibiotics
Dysentery	An inflammation of the colon characterized by multiple daily stool , low fever, and intestinal cramps	enteric man-to-man	None	Antibiotics and rehydration
Cholera	A serious acute intestinal disease characterized by vomiting, profuse watery stools, rapid dehydration and collapse	enteric man-to-man	Partial (Vaccine)	No specific treatment, rehydration procedure may be very effective
Hepatitis	An acute infectious disease followed by jaundice	man-to-man enteric	None	No specific treatment
Plague	A highly fatal infectious disease characterized by fever, prostration, pneumonia, and coma	vector borne man-to-man (pneumonic)	Partial	Antibiotics
Smallpox	An eruptive feverish disease characterized by extensive cutanous lesions	man-to-man	Vaccine	No specific treatment. (Antibiotics to control secondary infection)
Typhus	A generalized disease characterized by high fever	vector borne	Vaccine	Antibiotics
Whooping Cough	An infectious disease characterized by inflammation of the respiratory tract and peculiar paroxysms of cough	man-to-man	Vaccine	No specific treatment
Measles	An acute highly communicable viral disease characterized by a dusky-red blotchy rash	man-to-man	Vaccine	Gamma globulin
Diphtheria	An acute infectious disease of tonsils, pharynx, larynx or nose, and occasionally of other mucous membranes or skin	man-to-man enteric	Vaccine	Antidiphtheria serum
Gastroenteritis	Inflammation of the stomach and intestines characterized by severe diarrhea, dehydration and acidity	enteric	None	Antibiotics mildly effective, rehydration procedure may be very effective
Scarlet Fever	A streptococcal sore throat in which infectious agent produces a rash	man-to-man	None	Antibiotics
Meningococcal Meningitis	An acute bacterial disease characterized by sudden onset of fever, headache, nausea, and frequently a rash	man-to-man	None	Antibiotics

D. Non-Communicable and Chronic Disease

The approach to the problem of medical support postattack for those afflicted with non-communicable and chronic disease was analogous to that used for communicable disease. Life and death were considered rather than comfort, activity limitation, etc.; in addition, "Preventable Deaths" were calculated.

Table II shows an estimated preventable death rate from non-communicable and chronic disease of about 2 percent of the total surviving population. This is certainly small in comparison with the communicable disease threat.

Table II

PREVENTABLE DEATH RATES FOR MAJOR NON-COMMUNICABLE AND CHRONIC DISEASES UNDER POSTATTACK CONDITIONS

		A	B	C	D
		Expected Prevalence (1961-63 Rates Except for Tuberculosis 1928-31 Rates) (% of Total Population)	Preventable Fatality Rates = 1928-29 Case Fatality Rates less 1961-63 Case Fatality Rates (% of Prevalence)	Untreated Fatality Rates (% of Total Population)	Preventable Fatality Rates (Col. A x Col. B) (% of Total Population)
1	Diseases of the heart	7.079	24.0-5.2 = 18.8	1.700	1.331
2	Malignant Neoplasms	0.468	71.0-32.1 = 38.9	0.332	0.182
3	Diabetes	1.064	25.0-1.6 = 23.4	0.266	0.249
4	Vascular Lesions Affecting the Central Nervous System	0.930	20.0-11.0 = 9.0	0.186	0.084
5	Tuberculosis	0.472	16.3-1.5 = 14.8	0.077	0.070
6	Conditions of the Genitourinary System	2.203	3.3-0.7 = 2.6	0.073	0.057
7	Ulcer of the Stomach and Duodenum	1.080	3.2-0.6 = 2.6	0.035	0.028
8	Cirrhosis of the Liver	1.000	1.3-1.2 = 0.1	0.012	0.001
9	Asthma and Hay Fever	2.219	0.3-0.1 = 0.2	0.007	0.004
10	Diseases of the Bones and Organs of Locomotion	8.140	0.1-.02 = 0.08	0.008	0.007
Total		-	-	2.696	2.013

E. Conclusions

1. Communicable Disease

1) With current postattack medical preparedness measures, deaths will approximate 20 percent of the survivors. This might be as high as 25 percent or as low as 3 percent. The "preventable deaths" approximate 15 percent of the survivors.

2) The expected number of communicable disease fatalities is divided about 50 percent among those transmitted man-to-man, 5 percent vector borne, and 45 percent enteric, provided no additional preparedness measures are undertaken.[7/]

3) The calculations of preventable deaths indicate the relative effectiveness of preattack preparedness measures. Referring to Appendix G, it was concluded that:

a) Enteric diseases (gastroenteritis and dysentery) are very significant.

b) In large measure because enteric diseases are important, elementary countermeasures (personal hygiene and use of antiseptics) assume importance.

c) Considering one-at-a-time medical and public health measures, all are about equally effective. This conclusion relates to effectiveness and not to cost-effectiveness or to feasibility.

2. Non-Communicable and Chronic Disease

1) Fatalities from non-communicable and chronic disease will be slightly higher (2.5 to 3%) than they now are in the preattack environment of the United States. Thus, in terms of preventable fatalities, their importance is small.

2) Prevalence and fatality data from Table II indicate the relative unimportance of these in contrast with communicable disease.

F. Implication

In view of these conclusions the next Section deals with communicable diseases only. Specifically, it will deal with those determinants of postattack epidemics which can be influenced by preattack measures.

7/ Hallan, et al., op. cit.

III. EPIDEMIOLOGICAL CONSIDERATIONS FOR COMMUNICABLE DISEASES

A. Description of the Problem

As stated in the previous section, the majority of all preventable fatalities under late post nuclear attack conditions are likely to be due to communicable diseases.

For communitywide prevention or control of infectious diseases, programs can be divided into three categories:

1) Prevent the influx of infective individuals into the community through quarantine and elimination of the focus of infection by effective cure.
2) Decrease the transmission of pathogenic organisms between infective and susceptible individuals through hygiene and sanitation.
3) Decrease the susceptibility or increase in the resistance of individuals through prophylactic use of antibiotics or vaccination.

The problem to be discussed in this Section is how to build an infectious disease caseload generation model for estimating preattack cost and postattack benefit under alternative programs aimed against postattack communicable diseases. By permitting such a cost-benefit analysis, this model will be able to assist in the choice of preventive programs to be adopted. The following discussion is restricted to respiratory infections transmitted from man to man, but conceivably it can be extended to include fecally and vectorially transmitted infections.

b. Methodology

The Soper-Reed-Frost Model, generally accepted mathematical model of an infectious disease, was used. This model, restricted to infections transmitted from man to man, has two parameters: the contact rate λ and the host susceptibility aD. The model is independent of the community size N, insofar as the contact habits of an individual community member (as expressed in λ) are independent of N.

The values of the two parameters are determined by the infectious disease which they represent, by the community concerned, and by several behavioral and environmental factors including radiation. The magnitudes of the effects exerted by these determinants upon the two parameters were estimated from a literature survey for some communicable diseases of potential post nuclear attack importance. The expected parameter values can be estimated for the relevant post nuclear attack conditions by means of a statistical estimation of missing values based on literature values. Conceivably, parameter values (not found in literature) for other communicable diseases can be obtained by interpolation using a statistical model of homogeneity.

By using the estimated parameters, a mathematical model can be constructed to simulate those communicable diseases, and their attack rate--applicable to communities under similar behavioral and environmental conditions--that will be epidemic in the relevant post nuclear attack community. Also, various curative and preventive measures, either alone or combined, can be analyzed with the model, and their cost-effectiveness estimated.

C. The Mathematical Model of an Infectious Disease in a Community

The following model (by Soper[8], Reed and Frost[9]) of an infectious disease in a community is used throughout this Chapter. Figure 7[10] presents as compartments the three mutually exclusive states that are possible for any individual of the community at time t: susceptible (S_t individuals), infective (I_t individuals), or immune (Z_t individuals). The total number of individuals in the community at time t is: $N_t = S_t + I_t + Z_t$. Possible transitions in Figure 7 between states are indicated by arrows. Since the community is assumed to be isolated, N_t changes only by birth and death. The rates at which the transitions take place are given beside each arrow and are expressed in number of individuals per year.

As shown in the figure, a certain proportion of the infected persons becomes infective and each infective person remains in this state for a duration of D days before becoming immune.

8/ Soper, H. E. "Interpretation of Periodicity in Disease Prevalence," Journal of the Royal Statistical Society, Vol. 92, 1929, pp. 34-73.

9/ Reed, L. J. and W. H. Frost (1928), as quoted by Abbey in: "An Examination of the Reed-Frost Theory of Epidemics," Human Biology, Vol. 24, 1952, pp. 201-233.

10/ The definitions of the various symbols used in the model are summarized in the Glossary of Terms, Appendix A.

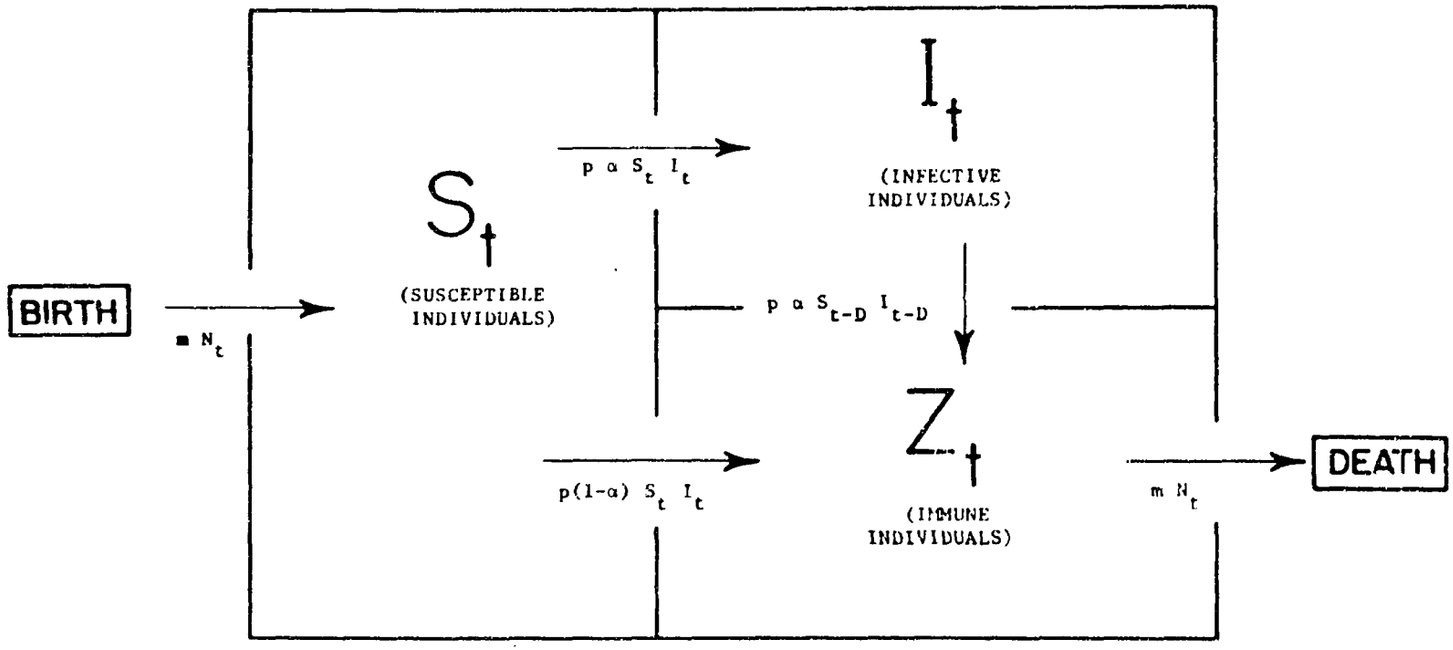


Fig. 7. Compartmental Transition Diagram of a Modified Soper Model of an Infectious Disease in a Community. (For Definition of the Symbols See Appendix A.)

In Figure 7, if there is a state of equilibrium ("steady state") so that the influx and outflux are equal for each compartment, the time subscripts can be omitted. Thus, in steady state, the left compartment has the following relationship:

$$Nm = ISp \ , \quad \text{or } m = \frac{IS}{N} p \ , \quad \text{or } m = \frac{I}{N} \frac{S}{N} pN \ ,$$

where

- m is the birth rate, expressed in the proportion of the community members that is being born per unit of time
- p is the probability of at least one contact between any two specified persons in the community per unit of time.

In steady state the transition between the left and the upper right compartment has the following relationship:

$$\frac{I}{D} = \alpha Nm \quad \text{or} \quad \frac{\frac{I}{N}}{\alpha D} = m \ ,$$

where α is the proportion of newly infected persons that will become infective. The last two equations make up the Soper model.

Reed and Frost refined the Soper model by taking into account the event that a susceptible individual may be infected more than once during the incubation period of the disease. The Soper-Reed-Frost model is as follows:

$$m(1 - e^{\frac{-\lambda i}{m}}) = s(1 - e^{-\lambda i}) = \frac{i}{\alpha D} \ , \qquad (2)$$

where

- e is the base of the Naperian (natural) logarithm with the constant value 2.71828...
- λ is the rate (number of persons per unit of time) at which a community member establishes contacts with the other susceptible, infective, or immune members. This rate is named the contact rate.
- i is the proportion of the community that is infective.

A major advantage of using λ instead of p is that with λ the variables and parameters are independent of size N of the community. The probability p of at least one contact between any two specified community members per time unit is replaced by λ, the rate at which a community member establishes contact with other members:

$$\lambda = p(N-1) \ . \qquad (3)$$

Likewise, the prevalences S and I are replaced with prevalence ratios s and i expressed as proportions of N.

According to Kermack and McKendrick[11] an epidemic will occur only when $s\alpha D\lambda > 1$. If this is the case, the value of the product $\alpha D\lambda$ can predict the crude attack rate when s, the proportion of susceptibles in the community, approaches the value of one (Table H-I, Appendix H). Thus, if the parameters are known, the modified model can be used to estimate the likelihood that an epidemic will occur and the size of the epidemic. The various methods of estimating these parameters for certain diseases from literature data are described in Appendix H.

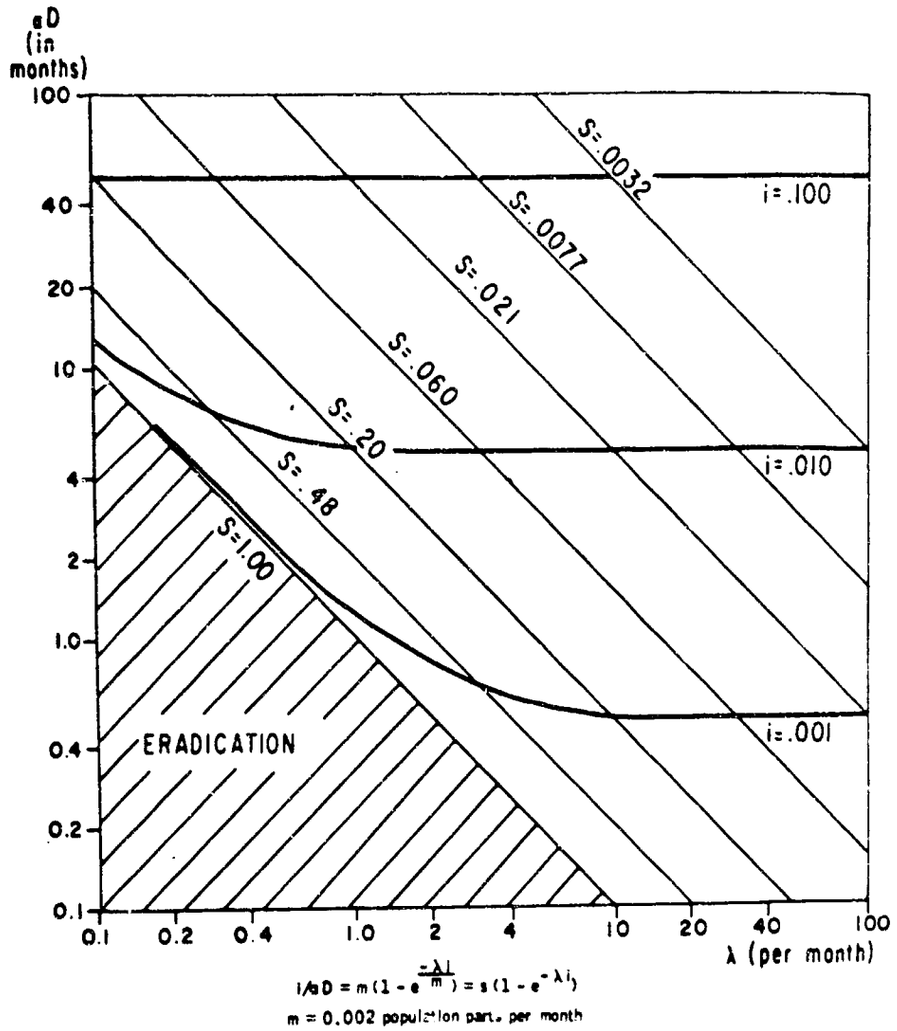
A nomogram of the Soper-Reed-Frost model (Figure 8) shows the sensitivities of the values of i for changes in the parameters λ and αD respectively. It follows from this nomogram that, according to current conceptual knowledge, the influence of the host susceptibility on the index i of disease and death is much greater than the influence of the contact rate.

The basic assumptions underlying the Soper-Reed-Frost model are summarized as follows:

1) The interval between infection and the beginning of infectivity ("incubation period" or "latent period") is constant.
2) Every person is susceptible (nonimmune), unless he has been infected previously; then he is immune.
3) Infection is spread by direct contact between infectives and susceptibles; there are no other contact possibilities. (Amplification: Current views in the bacteriological and virological literature (for instance Rammelkamp[12]) are in keeping with this assumption, which is limited to those infectious diseases which are transmitted from man to man by air droplet nuclei and confer complete immunity for life.)
4) After direct contact the susceptible becomes infective for a certain period of time; then he becomes nonsusceptible and noninfective.
5) Each person has a fixed probability of coming into contact with any other person within one incubation period; the probability is the same for any combination of two persons regardless of whether they are susceptible, infective, or immune.
6) The persons in the community are wholly segregated from persons outside the community.

11/ Kermack, W. O. and A. G. McKendrick. "A Contribution to the Mathematical Theory of Epidemics," Proceedings of the Royal Society of London, Series A, Vol. 115, 1927, pp. 700-721.

12/ Rammelkamp, C. H. "Epidemiology of Streptococcal Infections," Harvey Lectures, Vol. 51, 1955-1956, pp. 113-142.



Source: Voors, A. W.: A Modified Soper-Reed-Trust Model as a Guide to the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

Fig. 8. Nomogram of Susceptibles' (s) and Infectives' (i) Prevalence by Contact Rate (λ) and Non-Specific Human Susceptibility (αD) in a Deterministic Chain-Infection Model with an Influx of Susceptibles at a Constant Rate (m), for a Stationary and Stable Population with One Unique Age of Death Being (1/m).

D. Regression of the Soper-Reed-Frost Model Parameters on Several Behavioral and Environmental Characteristics.

1. Regression of the Contact Rate

Several behavioral and environmental attributes are likely to influence the level of the contact rate. The following nine attributes are considered: radiation, crowding within the household, size of the household, relative humidity, temperature level, temperature changes, season, ventilation and air pollution. (These attributes are described in more detail in Appendix I.) For the assessment of the effects as exerted by these attributes upon the contact rate, it is necessary to measure this contact rate. Within the household, the contact rate can be measured as secondary attack ratio and, in the community, the contact rate can be measured as crude attack rate. Both methods are applicable under the restriction that the persons at risk are known to be susceptible (Appendix H). Using data from the literature, the influence of these attributes on the contact rate were assessed (Appendix I). The results (Table III) indicate that only sudden temperature changes from warm to cold are likely to largely increase the rate of contact. Other factors increase the rate of contact, but only by a moderate or inconclusive magnitude.

Table III

CURRENT CONCEPTUAL KNOWLEDGE OF THE INFLUENCE OF NINE COMMUNITY ATTRIBUTES ON THE RESPIRATORY DISEASE CONTACT RATE

Community Attribute	Influence of the Attribute on the Contact Rate	
	Direction	Magnitude
Radiation	Increase	Inconclusive
Crowding within households	Increase	Small
Size of households	Increase	Inconclusive
Relative humidity	Decrease	Large
Temperature level of environment	Varying	Inconclusive
Temperature change of environment: Warm ⟶ Cold	Increase	Moderate
Season	Increase in Winter	Inconclusive
Ventilation	Decrease	Moderate
Air pollution	Increase	Inconclusive

2. Regression of Host Susceptibility

Several human-behavioral and environmental attributes are likely to influence the degree of host susceptibility. The following attributes will be considered: radiation, nutritional status, air pollution, emotional and genetic effects. For the assessment of the effects as exerted by these attributes upon the degree of susceptibility, it is necessary to measure this susceptibility. In the case of lifelong-immunity conferring disease in a stationary and stable population, non-specific host resistance can be derived from the prevalence of infectives (see Appendix H, Section III). Using data from the literature, the influence of the five attributes mentioned on the host susceptibility were assessed (Appendix J). The results (Table IV) indicate that radiation has an overwhelming influence on the host susceptibility.

Table IV

CURRENT CONCEPTUAL KNOWLEDGE OF THE INFLUENCE OF FIVE COMMUNITY ATTRIBUTES ON HOST SUSCEPTIBILITY

Community Attribute	Influence of the Attribute on the Host Susceptibility	
	Direction	Magnitude
Radiation	Increase	Large
Acute Starvation	Increase	Moderate
Air Pollution	Increase	Inconclusive
Emotional	Increase	Inconclusive
Genetically Increased Susceptibility	Increase	Inconclusive

3. Inferences Made from Observed Regressions Concerning One Respiratory Disease and Applicable to Others

a. Assumptions

The objectives of this paper seem to justify the use of some generalizations which are not accurate in detail but do yield information adequate for predictions and which should not be rejected because of imprecision, provided that these generalizations are internally consistent. It is desirable that they be tested using independently replicated data. So far, some observations

of respiratory diseases have been discussed which do not confer lifelong immunity or which are caused by organisms that do not have constant antigenicity (common cold, adenovirus 4, influenze type A). It would be advantageous to be able to apply the parameters obtained from these data to other respiratory diseases which do confer lifelong immunity and which are caused by organisms with more or less constant antigenicity (tuberculosis, diphtheria, meningococcosis, whooping cough, measles, German measles, mumps, chickenpox). The following four assumptions are necessary for application of the data:

1) During an epidemic, the effect of the organism on the contact rate must remain constant. According to the relevant literature, constancy of the prevailing strain during an epidemic has usually been observed in the case of influenza. For instance, Chu, Andrews, and Gledhill[13/] wrote about the 1948-1949 epidemic:

> "Rather to our surprise, almost all the strains from Western Europe, from Italy to Iceland, and one strain each from Canada and the U. S. A., were antigenically very homogeneous."

Furthermore, Jensen[14/] wrote:

> "Viruses collected from many parts of the world during an influenza season usually crossreact serologically with prototypic viruses of the year."

Later, Isaacs et al.[15/] wrote:

> "During the period 1957-1960 large numbers of influenza (type) A viruses were received at the World Influenza Centre from countries throughout the world. With one exception, all the strains were antigenically closely related to the A2 ("Asian" family) viruses isolated early in the Asian flu epidemic, and strikingly different from the A-prime ("A-prime" family) strains of the previous decade."

13/ Chu, C. M., C. H. Andrews, and A. W. Gledhill. "Influenza in 1948-1949," World Health Organization Bulletin, Vol. 3, 1950, pp. 187-214.

14/ "The Nature of Serological Relationships Among Influenza Viruses," op. cit.

15/ Isaacs, A., R. J. C. Hart, and V. G. Law, "Influenza Viruses, 1957-1960," World Health Organization Bulletin, Vol. 26, 1962, pp. 253-259.

In the case of other diseases, Webster[16/] measured the virulence of Pasteurella (in rabbits, chickens, and mice) and of mouse typhoid for various stages of the epidemics. "The results ... invariably ... showed a constancy and fixity of disease-producing power of a given strain of organisms under all conditions of natural infection ...".

2) For respiratory diseases (such as common cold and influenza) that do not confer lifelong immunity, the susceptibles and immunes are distributed at random within each age group with respect to characteristics like crowding and air temperature.

3) There is no interaction between any two of the following characteristics which affect the contact rate and the host susceptibility:

a) Host resistance.

b) Host behavior (like crowding).

c) Environmental characteristics (like relative humidity, temperature change, air pollution, ventilation).

d) Organism characteristics.

For the pertinence and validity of this assumption, see Appendix K.

4) The effects of community and disease on the contact rate are such that there is statistical homogeneity (independent effects). The same holds for their effects on the host susceptibility. The pertinence and validity of these assumptions are discussed in Appendix K.

There is a multiplicity of factors (Voors[17/]) which permits the assumptions of random distribution and interaction. The assumption of no interaction is testable by repeated independent observations.

A possible exception is the interaction between factors (c) and (d); the importance of such an interaction is discussed in Appendix I, Section 4. Other possible exceptions are the interactions between effects (a) and (d) and between (b) and (d): Langmuir,[18/] quoting

---

16/ Webster, L. T. "Experimental Epidemiology," Medicine, Vol. 25, 1946, pp. 77-109.

17/ Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

18/ Langmuir, A. D. "Epidemiology of Air-Borne Infection," Bacteriological Reviews, Vol. 25, 1961, pp. 173-181.

Lurie[19] and Hatch,[20] points out that droplets of 5 microns or less are necessary in the transmission of tuberculosis while many respiratory diseases are transmitted by larger droplets. This suggests interaction between the organism factor and both the host resistance and human behavioral factors effect which affects the contact rate. In spite of these exceptions, the assumption of no interaction will be maintained for the reasons mentioned in the beginning of this subsection.

b. Conclusions Concerning Regression of Contact Rate

In view of the assumptions concerning the regression of the contact rate of respiratory diseases on environmental and behavioral factors, the following conclusions can be drawn:

1) The lower the relative humidity indoors, the higher the contact rate (marked influence).
2) The more extreme the temperature changes to which the individuals are subjected, the higher the contact rate (medium influence).
3) The more crowded the household of susceptible persons, the higher the contact rate (small influence).
4) The higher the ventilation in the enclosed area containing a group of people, the lower their respiratory contact rate (medium influences).

c. Conclusions Concerning Regression of Host Susceptibility

Host susceptibility is increased by the following factors:

1) Radiation (marked influence).
2) Malnutrition (slight influence under postnuclear attack conditions).
3) Air pollution (possibly a slight influence).

19/ Lurie, M. B., et al. "An Evaluation of the Method of Quantitative Air-Borne Infection and Its Use in the Study of the Pathogenesis of Tuberculosis," American Review of Tuberculosis, Vol. 61, 1950, pp. 765-797.

20/ Hatch, T. F. "Distribution and Deposition of Inhaled Particles in Respiratory Tract," Bacteriological Reviews, Vol. 25, 1961, pp. 237-240.

E. Assessment of Contact Rate for Some Respiratory Diseases of Post-Shelter Importance

1. Assessment of the Contact Rate for Asian Influenza

Literature data concerning the Asian influenza epidemic of 1957-1958 were obtained for various communities. Using the method described in Appendix H and assuming that $\alpha = 1/52$ year, one can arrive at values from these literature data for contact rate under different circumstances. [Amplification: The assumption that $D = 1/52$ year is arrived at in the following way. According to Gordon[21], the average duration of infectivity is 7.5 days; hence, if the degree of infectivity is equally distributed over these days the mean point of infectivity is $(7.5)/2 = 3.75$ days after its beginning. Jordan et al.[22] found the average interval between successive cases to be 5.3 days. Assuming an average incubation period of 2 days (Gordon[21], Woodall et al.[23]), this would imply that the mean point in the duration of infectivity is at $5.3 - 2.0 = 3.3$ days after its beginning. Since the figures 3.75 and 3.3 show a fair agreement, 7 days or about 1/52 year is used as "duration of infectivity" for assessment of contact rate]

An example of the calculations involved is as follows: In a cottage at a children's institution, Bell et al.[24] found that out of 34 girls aged 9-12 years, 18 became infected. Thus, the primary attack rate is $18/34 = .53$. According to D. G. Kendall's table[25] the corresponding value of αD is 1.40. Since $\alpha D = 1/52$, $\alpha = 1.40 \times 52 = 73$ per year.

The results of these contact calculations applied to suitable literature data are given in Table V.

21/ Gordon, J. E. (ed.) Control of Communicable Diseases in Man (9th edition), American Public Health Association. New York, 1960.

22/ Jordan, W. S., et al. "A Study of Illness in a Group of Cleveland Families, XVII. The Occurrence of Asian Influenza," American Journal of Hygiene, Vol. 68, 1958, pp. 190-212.

23/ Woodall, J., K. C. K. Rowson, and J. C. McDonald. "Age and Asian Influenza," British Medical Journal, No. 5108, Vol. 2, 1958, pp. 1316-1318.

24/ Bell, J. A., et al. "Epidemiological Observations on Two Outbreaks of Asian Influenza in a Children's Institution," American Journal of Hygiene, Vol. 73, 1961, pp. 84-89.

25/ See Appendix H, Table H-1.

Table V

CONTACT RATES[a] FOR ASIAN INFLUENZA BY AGE DURING THE PANDEMIC OF 1957-1958, IN DIFFERENT COMMUNITIES

City or State	Type of Community	Race	Social Class	Contact Rates by Age (in Years)					
				3-5	6-8	9-9	9-12	13-17	20-50
D. C. [d]	Children's Institution	Negro	Lower	208	182	83	F. 73 M. 94	F. 67 M. 72	
La. [e]	Day School	Negro	Lower	73	83			94	
La. [e]	Day School	Negro	Lower	57	83			88	
La. [e]	Day School	Negro	Lower	68	78			88	
La. [e]	Day School	White	Middle & High	57	57			62	
Ohio [f]	Families	White	Middle & High	89					
Mo. [g]	High School Families	White	Middle & High	49[b]					
				33[b]					
Melbourne [h]	Individuals	White		89					
Liverpool [i]	Remand Home	White	Lower		99				
Liverpool [i]	Temporary Accomodation	White	Middle	68					
London [j]	Physician's Practice (Families)	White	Lower	18[b,c]					
England [k]	Air Force Boarding School	White	Higher					98	

a/ Expressed per year from 1957 to 1958; calculated from literature sources listed below.

b/ Clinical diagnosis without serological confirmation.

c/ Calculated from secondary attack rate instead of primary.

d/ Source: Bell, J. A., et al. "Epidemiological Observations on Two Outbreaks of Asian Influenza in a Children's Institution," American Journal of Hygiene, Vol. 73, 1961, pp. 84-89.

e/ Source: Dunn, F. L., et al. "Epidemiological Studies of Asian Influenza in a Louisiana Parish," American Journal of Hygiene, Vol. 70, 1959, pp. 351-371.

f/ Source: Jordan, W. S., et al. "A Study of Illness in a Group of Cleveland Families. XVII. The Occurrence of Asian Influenza," American Journal of Hygiene, Vol. 68, 1958, pp. 190-212.

g/ Source: Chin, T. D. Y., et al. "Morbidity and Mortality Characteristics of Asian Strain Influenza," Public Health Reports, Vol. 75, 1960, pp. 149-158.

h/ Source: Krough, E. V., et al. "A Serologic Survey of the Epidemic of Asian-Type Influenza in Melbourne, 1957," American Journal of Hygiene, Vol. 68, 1958, pp. 1-5.

i/ Source: Schwartz, K. and W. H. Parry. "A Study of Asian Influenza Epidemiology and Control Measures in Liverpool Children," Medical Officer, Vol. 99, 1958, pp. 59-61.

j/ Source: Woodall, J., K. C. K. Rowson, and J. C. McDonald. "Age and Asian Influenza," British Medical Journal, No. 5108, Vol. 2, 1958, pp. 1316-1318.

k/ Source: Owen, I., et al. "Asian Influenza in a R. A. F. Boy Entrants School," Public Health, Vol. 72, 1958, pp. 134-142.

2. Assessment of Contact Rate for Tuberculosis, Measles, Whooping Cough, and Diphtheria

When an immunity producing disease is transmitted in a community which is in a steady demographical state and has a constant influx of susceptibles, the model predicts that the prevalence of susceptibles and infectives will each fluctuate over time around a certain equilibrium value: after a maximum of infectives, the concomitant exhaustion of susceptibles has to be built up again by the influx of new susceptibles before a new maximum ("epidemic peak") of infectives can be expected. The "delay time" t in the start of a new epidemic peak since the end of a previous one has a value $\tilde{t}$ of maximum likelihood (Appendix H).

$$\tilde{t} = \frac{s_o}{m}$$

where s_o is the equilibrium value for the prevalence of susceptibles in the community, expressed as a proportion of the community size and m is the birth rate.

Since each of the three characteristics in this equation can be observed, this equation constitutes a test for the realism of the model. Values for t can be observed by studying time series of infectives (or by studying time series of an index of infectives like cause-specific mortality), either through assessment of time lag for maximal autocorrelation (Table VI) or through assessment of the average time interval of consecutive crossings between the observed time series and its nonlinear regression line (Table K-IV). Observed values for s_o and m are available in the literature.[26/] The observed and expected values are in fair agreement (Table VII) and this tends to support the realism of the assumptions underlying the model.

Therefore, by assuming values for αD from current literature and by using the formula $\lambda = \frac{1}{s_o \alpha D}$, the contact rate can be estimated (Table VIII).

26/ Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

Table VI

'S (IN YEARS) FOR MAXIMAL AUTOCORRELATION IN TIME SERIES OF REPORTED DEATHS

Four Infectious Diseases in 19 Large United States Cities, 1901-1949[a/,b/,c/]

Cities	of Infectious Respiratory Diseases			
	Tuberculosis	Measles	Whooping Cough	Diphtheria
Baltimore, Md.	30	3	4	9
Boston, Mass.	30	3	3.5	10
Chicago, Ill.	42 or 13	2	3.5	9
Cleveland, Ohio	--	3	4	7 or 14
Columbus, Ohio	--	2.5	2	5 or 10
Indianapolis, Ind.	21	3	3	5 or 10
Kansas City, Mo.	--	3	3	12
Los Angeles, Calif.	15	3.5	2.5	10 or 15
Louisville, Ky	24	2.5	2.5	6.5 or 13
Milwaukee, Wis.	36	3	3.5	7 or 14
Minneapolis, Minn.	--	2.5	2.5	5 or 10
New Orleans, La.	26	3	3	6.5 or 13
New York City, N. Y.	21	2	3.5	4 or 10
Philadelphia, Pa.	--	3	4	10
Pittsburgh, Pa.	--	2.5	2	6.5 or 13
Providence, R. I.	36	3	4	13
Saint Louis, Mo.	26	3.5	3	6 or 12
San Francisco, Calif.	44	4	5.5	7 or 14
Washington, D. C.	28	3	3	9
Average Lag Duration	27.5 or 28.0	2.89	3.26	7.76 or 11.58

a/ In the case of tuberculosis the observation period was up to 150 years.

b/ The lags were estimated by eye from autocorrelograms. In case of uncertainty of choice between two values, both values were entered.

c/ Source: Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

Table VII

PREDICTIONS AND OBSERVATIONS CONCERNING THE MODE OF DURATION OF THE INTERVAL BETWEEN EPIDEMIC WAVES AND THE AVERAGE DURATION OF THE INTERVAL BETWEEN EPIDEMIC PEAKS

Four Infectious Diseases in 19 Large United States Cities, 1901-1949[a/]

Diseases	Prevalence of Susceptibles	Duration of Intervals in Years		
		Predicted by Assessing[b/] s_o and m	Observed From Curve Crossings Between Epidemic Waves and Their Regression Line[c/]	Observed From Autocorrelogram Interpretation[d/]
Measles	.0762	3.8	3.4	2.9
Whooping Cough	.0906	4.5	3.4	3.3
Diphtheria	.2451	12.3	5.1	7.8 or 11.6
Tuberculosis	.2963	>17.1	13.7	27.5 or 28.0

a/ Source: Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

b/ Predicted value = $\frac{s_o}{m}$

c/ See Table V of Appendix K.

d/ See Table VI.

Table VIII

CALCULATION OF POINT ESTIMATES FOR CONTACT RATE PRIOR TO THE ANTIBIOTIC ERA

Diseases	Expectancy of Duration of Infectivity αD [a/] (days)	Prevalence of Susceptibles s [b/] (proportion)	Contact Rate $\lambda = \frac{1}{s \alpha D}$ (per day)
Diphtheria	10	.2451	.41
Whooping Cough	21	.0906	.53
Measles	9	.0762	1.46
Influenza	3	.2000	1.67

a/ Source: Gordon, J. E. (ed.) Control of Communicable Diseases in Man, American Public Health Association, 9th edition. New York, 1960.

b/ Source: Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

F. Assignment of Independent and Dependent Variables to the Disease Caseload Generation Subroutine

1. Independent Variables

Independent variables for the disease caseload generation subroutine should include alternatives in either post nuclear attack circumstances or preparedness measures which have a marked influence on post nuclear attack survival.

Radiation dose is a major determinant of survival. In keeping with current literature, it is assumed that the subjects have been irradiated by a single dose of 200r.[27/]

Other major determinants of survival are: adequate supplies of essentials like food and protection against climate exposure (viz., mortality in the Netherlands during World War II[28/]). It is assumed that these supplies will be sufficient.

It is also assumed that the following measures have been taken:

1) Effective control of rodents and arthropods.[29/]
2) Equal health service and facilities for all social classes if uniformly adequate service cannot be maintained.
3) Avoidance of the undue concentration of the more susceptible persons into a "shelter hospital", that would act as a focus of infection.
4) Avoidance of mass prophylaxis with antibiotics that would cause drug resistance.
5) Avoidance of disrupting social conditions.

27/ Stoner, R. D., M W. Hess, and V. P. Bond. Radiation and Infection: An Annotated Bibliography. Upton, N. Y.: Medical Research Center, Brookhaven National Laboratory, May 1965.

28/ Netherlands' Ministry of Social Affairs: Malnutrition and Starvation. Edited by G. C. E. Burger, J. C. Drummond, and H. R. Sanstead. The Hague: General State Printing Office, 1948.

29/ Johnson, T. and D. R. Johnston. Vectorborne Disease and Control, Final Report, R-OU-303. Research Triangle Park, N. C.: Research Triangle Institute, June 1968.

2. Dependent Variables

The major decisions for post nuclear attack health preparedness can (under the above assumptions) be categorized as indicated in the beginning of this Chapter:

1) Prevention of the influx of infective individuals into the community by quarantine and/or elimination of the focus of infection by effective cure of the infective individuals.
2) Decrease in the transmission of pathogenic organisms between infective and susceptible individuals through hygiene and sanitation.
3) Decrease in the susceptibility or increase in the resistance of individuals through vaccination or prophylactic antibiotics.

Thus, the decision model includes the following alternatives:

1) Quarantine or no quarantine (various degrees of quarantine would not be relevant; see Appendix L).
2) Increased or non-increased level of hygiene and sanitation.
3) Postattack vaccinations and mass prophylaxis with antibiotics: yes or no.

Thus, there are $2^3 = 8$ possible alternative combinatorial sets of parameter values (Table IX).

6. Implications of the Soper-Reed-Frost Model[30/]

A mathematical model of a respiratory infection in a community has been developed consistent with current infectious disease theory. This model can be used to provide predictions--most likely in view of the state of the art--concerning epidemics under unknown future conditions like atomic attack or food shortage.

30/ Soper, op. cit., pp. 34-73, and Reed and Frost, op, cit., pp. 201-233.

Table IX

ALTERNATIVE SETS OF PARAMETER VALUES RESULTING FROM DECISIONS FOR THE DISEASE CASELOAD GENERATION MODEL[a/]

Type of Decision		
Quarantine	Hygiene and Sanitation	Vaccination or Antibiotics
-	-	-
-	-	+
-	+	-
-	+	+
+	-	-
+	-	+
+	+	-
+	+	+

a/ - decided not to use for prevention or control.

\+ decided to use for prevention or control.

IV. SUBMODEL INPUT AND OUTPUT DATA

A. General

This submodel describes all the essential elements of a series of simultaneous postattack epidemics in a finite geographical area, such as a community. The postattack results are described in terms of number of fatalities, number of patients diseased, amount of drugs used, and number of person-days of medical staff occupied.

Figure 9, Basic Inputs and Outputs of the Diseases and Chronic Conditions Submodel of the Total Emergency Health Care System Model, summarizes the submodel's inputs and outputs. These are discussed in detail in the following sections.

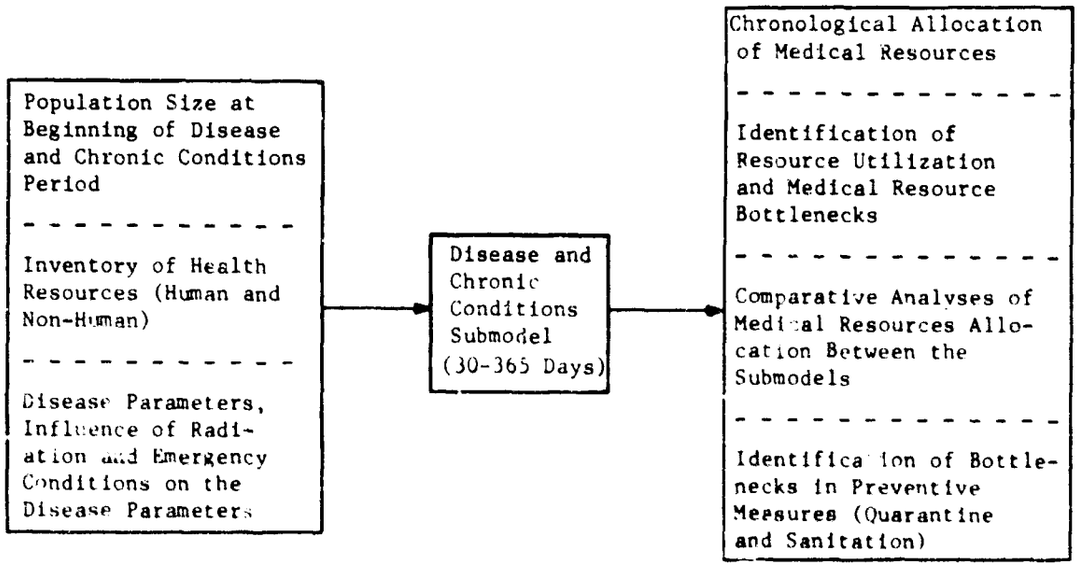


Fig. 9. Basic Inputs and Outputs of the Disease and Chronic Conditions Submodel of the Total Emergency Systems Model.

B. Input Data

1. Preattack Characteristics of the Communicable Diseases under Consideration

The various relevant characteristics as observed under preattack conditions, of the respective communicable diseases are manually entered for each disease separately into input cards. Then characteristics, values of which were obtained through a literature review, are:

1) The fraction of the population that is susceptible under preattack equilibrium conditions.
2) The fraction of the population that is infective under preattack equilibrium conditions.
3) The contact rate under preattack conditions.
4) The "Infective ratio" under preattack conditions, that is the fraction of all persons infected, that becomes infective in turn.
5) The average duration of infectivity in the infective persons under preattack conditions.
6) The case fatality rate under preattack conditions as applied to the infectives.

2. Effects of the Attack on the Disease Characteristics

Under the assumption that the attack will affect each of the selected communicable diseases to a similar extent, the following four coefficients are entered as input and serve as multiplication factors for all of the communicable diseases: (a) a coefficient for the infective ratio; (b) a coefficient for the contact rate; (c) a coefficient for the population fraction that is initially infective and (d) a coefficient for the population fraction that is initially susceptable. The values for these characteristics were extracted from the limited amount of related literature that was available and are entered manually as input.

3. Population Size

The size of the population at the beginning of the Disease and Chronic Conditions period as generated by the Immediate Effects Submodel, is entered manually as input.

4. Availability of Professional Staff and Drugs

The level of availability of professional staff and drugs are entered manually as input. Under the follow-on contract it is intended to model this level as a function of daily workload and stockpile depletion. It is also hoped that due consideration can be given in future research to priority decision rules regarding the respective degrees to which the various diseases are vitally important.

C. Output Data

Output from the submodel includes:

1. The number of newly infectives and the number of fatalities, both expressed by 5-day periods as fractions of the total population, for each of the diseases listed in Table I.
2. Drug requirements for each of the four drugs listed in Appendix G are printed as 5-day totals and as cumulative totals.
3. A combined listing and graphic plot of the total number of infectives for all diseases and the corresponding number of physicians required to treat these infectives. This information is given for each fifth day.
4. Final totals for the entire 365 day period covered by the model are computed for infectives (overall total and subtotals by disease), fatalities (overall total and subtotals by disease) drug requirements, and physician time requirements.

Details concerning the precise use of input cards and a sample of output format for the Disease and Chronic Conditions Submodel are presented in Appendix D and Chapter 4.

V. SUBMODEL FUNCTIONS AND LOGIC

A. General

The purpose of this section is to show the basic information flow through the Disease and Chronic Conditions Submodel and to describe the logic with which this submodel simulates the generation, treatment, and fate of the diseased persons.

B. Functions of the Submodel

The Disease and Chronic Conditions Submodel is designed to perform the following functions with respect to each of the pertinent diseases:

1. Predict tomorrow's fractions of newly infected and newly cured individuals on the basis of today's population fractions of susceptibles and infectives. This process begins 30 days after the attack and continues on a day-by-day basis for 365 days--always using yesterday's predicted fractions as today's given fractions.
2. Compute, for the obtained daily fraction of infectives, the medical staff and supplies consumed under assumed levels of availability; compute the respective fractions of fatality thus entailed; and record these results by five day periods.
3. Repeat this procedure for various levels of radiation (affecting the individual's susceptibility to disease and hence the rate of infection), for various levels of quarantine (eliminating some of the more esoteric diseases from consuming staff and supplies and from contributing to fatality), and for various levels of sanitation (affecting the rate of infection).

C. Submodel Logic

1. Data Flow

A step-by-step description of the flow chart for the Disease and Chronic Conditions Submodel (see Figure 10) is presented below.

Step 1. Control card parameters are read in. These parameters indicate the severity of radiation, the presence of quarantine, the level of sanitation measures, the vaccination-and-pretreatment status of the population, and the initial size of the population.

Step 2. Read in the Disease Table. The contents of this table were previously discussed under Section IV. B. 1.

Step 3. Set the time clock to indicate the first day to which the submodel is applied--actually the 31st day of the postattack period.

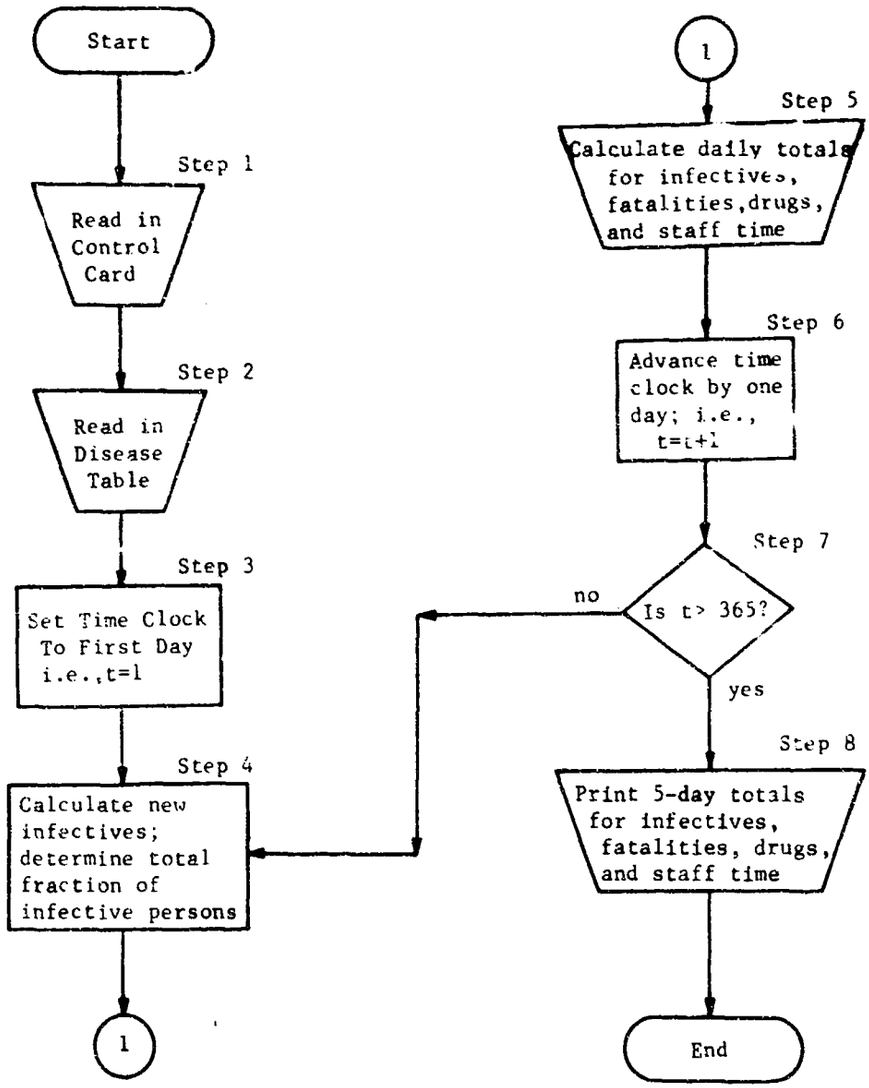


Fig. 10. Disease and Chronic Conditions Submodel Flow Chart

Step 4. Calculate for each disease the fraction of new infectives for the day on which the time is set. This current day's fraction of newly infectives is the product of (a) the last day's fraction of susceptibles, (b) the infection ratio, (c) the contact rate, and (d) the last day's fraction of infectives. The total fraction of infected persons is then determined by:

a) Subtracting the fraction of the previous day's case fatalities from the previous day's fraction of infectives. This result is called the "remainder of the previous day's infectives fraction."

b. Adding the current day's fraction of new infectives to the "remainder of the previous day's infective fraction"

c. Computing the fraction of cured infectives for the current day; i.e., the fraction of the population that passed from the infective into the immune state.

d. Subtracting the result of (c) from (b)

Step 5. Print output as previously discussed under Output Data; Section IV. C.

Step 6. The time clock is advanced by one day.

Step 7. A check is made for the end of the 365-day period to which this submodel is applied.

Step 8. Print final totals and subtotals as previously discussed under Output Data, Section IV. C.

2. Decision Rules

Two decision rules used in the Disease and Chronic Conditions Submodel are: (1) change in basic disease parameters and (2) factors relating to the increase of death rate due to the lack of physician time and/or drugs. The changes in disease parameters for fallout are described in Chapter 4 and factors relating to changes in death rate due to the insufficiency of medical resources is described in Appendix G.

It must be noted that this model is limited (a) by its consideration of each disease independently of the other diseases and (b) by disregarding the state in which each person finds himself with respect to the other diseases; i.e., susceptible, infective, immune-or-cured, or dead.

Chapter 4

Total Emergency Health Care System Model Case Study

I. INTRODUCTION

In consultation with Office of Civil Defense (OCD) and Public Health Service (PHS) personnel, New Orleans was chosen as the test city for the Total Emergency Health Care System Model. The availability of medical resource data and the inclusion of New Orleans in a recent damage assessment study were the prime reasons for its selection.

The weapon effects were from a 1.5 MT weapon, surface burst at a distance of approximately 9 miles south of the center of the city, which has a resident population of 1,002,000.

II. IMMEDIATE EFFECTS SUBMODEL

A. Input Data

1. Injuries

Since the geographic location of physicians (described below) was available only by Post Office ZIP Code areas, these areas were taken as the grids used in the model. For the weapon described above, the injury spectrum for each ZIP Code area was obtained through application of the unclassified Dikewood casualty curves.[1/] These curves indicate the percent of the population that will receive injuries of specified types as a function of shielding and weapon parameters. Used in the New Orleans case study were the Dikewood "brick building" curves (most appropriate for the area) and the overpressures calculated using the slant range from the weapon to each of the areas. Results of these analyses were prepared in the format described in Appendix D for processing by the sub-model. The 25 injuries studied and their treatment parameters are shown in Table X. It is noted that the treatment levels indicated for the injuries in this case study are generally higher than the preferred treatment levels described in Appendix B; i.e., "upgraded". A preliminary run of the New Orleans case study

---

1/ Davis, L. Wayne, et al. Prediction of Urban Casualties and the Medical Load From a High-Yield Nuclear Burst. Albuquerque, N. Mex.: The Dikewood Corporation, December, 1967.

Table X

INJURIES AND TREATMENT PARAMETERS
(New Orleans Case Study)

Injury	Description	Treatment Priority [1]	Golden Period (Hours) [2]	Medical Treatment Package [3]	Treatment Level [4]	Treatment Time (Hours)	Probability of Death With Treatment	Probability of Death Without Treatment
1	Laceration and open wound of back	9	6.00	W 10	3	0.50	0.00	0.05
2	Laceration and open wound of elbow, forearm, wrist	9	6.00	W 10	3	0.50	0.00	0.05
3	Laceration and open wound of face and neck	9	6.00	M 6	3	0.00	0.00	0.05
4	Dislocation of shoulder	9	6.00		1	0.00	0.00	0.00
5	Dislocation of wrist	9	4.00		1	0.50	0.00	0.00
6	Head concussion	9	12.00	H 11	3	0.50	0.00	0.10
7	Fracture of radius and/or ulna	9	24.00	C 5	3	0.50	0.03	0.10
8	Rib fracture	9	6.00	T 3	3	0.75	0.08	0.10
9	Pelvis fracture	6	6.00	P 4	2	2.00	0.10	0.75
10	Foreign body through orifice, pharynx and larynx	3	6.00	T 3	2	1.00	0.25	0.95
11	Eye and orbit laceration	9	6.00	W 10	3	0.75	0.20	0.25
12	Brain contusion	3	12.00	H 11	3	0.50	0.20	0.75
13	Chest and abdomen	4	6.00	T 3	2	2.50	0.25	0.95
14	Severe blast and moderate thermal	8	6.00	BA 8	2	4.00	0.25	0.70
15	Kidney injury	2	6.00	P 4	2	2.25	0.05	0.90
16	Severe blast and severe thermal	9	6.00		1	0.00	0.95	0.95
17	Skull fracture	6	12.00	H 11	3	1.00	0.35	0.90
18	Fracture of vertebra	6	12.00	V 16	2	2.25	0.25	0.95
19	Heart or lung injury	5	6.00	T 3	2	2.70	0.25	0.95
20	Moderate blast and moderate thermal	9	6.00	A 2	3	0.50	0.30	0.35
21	Burns over less than 10% of body	9	6.00	BA 8	3	0.25	0.00	0.01
22	Burns over 10-20% of body	9	6.00	BI 1	3	0.25	0.10	0.15
23	Burns over less than 10% of body LD_{50} Dose	9	96.00	BA 8	3	0.25	0.60	0.70
24	Radiation dose - LD_{50}	9	96.00	S 5	4	0.00	0.50	0.50
25	Radiation dose - LD_{75}	9	96.00	S 5	4	0.00	0.75	0.75

[1] Treatment Priority ranges from 1 to 9, with 1 being highest priority (See Appendix B).

[2] The period immediately following the attack in which delay in treatment does not cause the probability of death to rise.

[3] See Appendix M for the composition of Medical Treatment Packages.

[4] Codes 1, 2, 3, and 4 refer to no-treatment, surgical teams, physicians, and allied medical personnel, respectively.

indicated that the majority of the injuries were relatively minor. As a result, the available physician time was not being utilized when treatment levels defined in Appendix B were used. Therefore, treatment levels were upgraded to more realistically simulate the case study postattack situation.

The numbers of casualties determined for each of the 24 ZIP Code areas by type of injury are shown in Table XI. The Dikewood injury curves do not indicate possible multiple injuries per individual; the Total Emergency Health Care System Model treats each injury as an independent casualty. The grand total of casualties in Table XI indicates that 387,030 people (approximately 39 percent of the one-million preattack population) were injured by the weapon, either by fallout and/or direct effects. It is noted that most of the casualties suffered from minor lacerations and dislocations, moderate burns, and fallout radiation. In fact, fallout radiation accounted for 286,000, or 73 percent of all casualties.

2. Medical Resources

The preattack population (1,002,000) and the number of preattack physicians (2,076) located in the 24 ZIP Code areas of New Orleans were based on 1966 estimates furnished by the National Resource Analysis Center (NRAC). The physician data were based on the address at which the physician received his mail from the Federal Government. The number of allied medical personnel (6,585) in the entire city was obtained from a Public Health Service document;[2/] unfortunately, this information was not available by ZIP Code area.

Information on preattack medical supplies for New Orleans, supplied by the Public Health Service, was used to form treatment packages (items necessary for the treatment of specific injuries) for the case study. Requirements for these medical packages (described in Appendix M) and other treatment parameters (described in Appendix B) are shown in Table X for each of the 25 injuries.

Table XII shows the number of surviving physicians, allied medical personnel, and the number of "treatment packages" available to the physicians. These data on surviving physicians in each ZIP Code area were supplied by the Office of Civil Defense. The other medical resources (allied medical personnel and supplies) were assumed to survive the attack and to be distributed in the same proportion as surviving physicians. Under this assumption, 62 percent of the preattack resources survived and were distributed to the 24 ZIP Code areas.

2/ Public Health Service, Health Manpower Source Book, Section 19, Location of Manpower in 8 Occupations. Public Health Service Publication Number 263 (Section 19), Washington, D. C.: U. S. Government Printing Office, 1965.

Table XI

NUMBER OF INJURIES BY INJURY NUMBER
(In Tens)

INJURY NUMBER

ZIP CODE	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
20071	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20072	74	74	75	31	31	0	0	0	0	1	1	1	1	1	1	0	0	0	0	74	156	17	10	585	585
20073	56	56	57	25	25	0	0	0	0	1	1	1	1	1	1	0	0	0	0	56	133	15	0	0	0
20078	29	29	31	19	19	9	0	0	0	2	2	1	1	1	1	0	0	0	0	29	79	9	10	95	95
20082	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20872	247	247	256	156	156	55	0	0	0	9	9	6	4	4	4	2	1	1	0	247	642	71	290	320	320
20094	30	30	30	16	16	0	0	0	0	1	1	0	0	0	0	0	0	0	0	30	55	6	0	0	0
20112	17	17	18	10	10	0	0	0	0	0	0	0	0	0	0	0	0	0	0	17	35	4	0	135	135
20113	122	122	124	70	70	0	0	0	0	1	1	1	1	1	1	0	0	0	0	122	249	28	110	820	820
20114	217	217	248	146	146	133	5	2	13	31	31	10	9	9	9	1	12	1	11	217	538	60	10	605	605
20116	111	111	113	65	65	0	0	0	0	1	1	0	0	0	0	0	0	0	0	111	225	25	0	945	945
20117	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	143	16	10	2074	2074
20118	45	45	45	30	30	0	0	0	0	0	0	0	0	0	0	0	0	0	0	45	114	13	50	310	310
20119	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	98	10	90	1710	1710
20121	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20122	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	65	7	0	1680	1680
20123	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20124	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	175	175
20125	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	47	5	210	840	840
20126	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1130	1130
20127	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	175	175
20128	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	50	50
20130	80	80	81	58	58	0	0	0	0	1	1	1	1	1	1	0	0	0	0	80	147	19	20	500	500
20115	213	213	217	122	122	0	0	0	0	5	5	2	2	2	2	0	0	0	0	213	438	49	540	1285	1285
TOTAL	1241	1241	1295	749	748	197	5	2	13	53	53	23	20	20	20	3	13	2	11	1241	3194	354	1350	13434	13434

TOTAL FOR ALL INJURIES = 38703 (i.e., 387,030 injuries)

Table XII

MEDICAL PERSONNEL AND TREATMENT PACKAGES IN EACH ZIP CODE AREA

ZIP CODE	PHYSICIAN.	AUD. MED. PER.	BI	A	T	P	S	M	BA	W	H	V
70001	78.	248.	87	11	77	48	16	601	8971	225	14916	46
70032	1.	3.	1	0	0	0	1	7	115	2	191	0
70043	1.	3.	1	0	0	0	1	7	115	2	191	0
70053	6.	19.	6	0	5	3	8	46	690	17	1147	3
70059	7.	22.	7	1	6	4	10	53	805	20	1338	4
70062	6.	19.	6	0	5	3	8	46	690	17	1147	3
70067	2.	6.	2	0	1	1	2	15	230	5	382	1
70072	17.	54.	19	2	16	10	25	131	1955	49	3250	10
70075	0.	0.	0	0	0	0	0	0	0	0	0	0
70085	0.	0.	0	0	0	0	0	0	0	0	0	0
70094	7.	22.	7	1	6	4	10	53	805	20	1338	4
70102	0.	0.	0	0	0	0	0	0	0	0	0	0
70104	0.	0.	0	0	0	0	0	0	0	0	0	0
70105	0.	0.	0	0	0	0	0	0	0	0	0	0
70107	2.	6.	2	0	1	1	2	15	230	5	382	1
70109	0.	0.	0	0	0	0	0	0	0	0	0	0
70110	0.	0.	0	0	0	0	0	0	0	0	0	0
70112	104.	331.	116	15	103	64	155	801	11961	301	19888	62
70113	8.	25.	8	1	7	4	11	61	920	23	1529	4
70114	4.	13.	4	0	3	2	5	30	460	11	764	2
70115	500.	1590.	562	74	499	309	748	3853	57507	1447	95616	300
70116	25.	79.	28	3	24	15	37	192	2875	72	4780	15
70117	6.	19.	6	0	5	3	8	46	690	17	1147	3
70119	139.	442.	156	20	138	86	208	1071	15986	402	26581	83
70119	80.	254.	89	11	79	49	119	616	9201	231	15298	48
70120	2.	6.	2	0	1	1	2	15	230	5	382	1
70121	110.	350.	123	16	109	68	164	847	12651	318	21035	66
70122	14.	45.	15	2	13	8	20	107	1610	40	2677	8
70123	11.	35.	12	1	10	6	16	84	1265	31	2103	6
70124	24.	76.	26	3	23	14	35	184	2760	69	4589	14
70125	34.	108.	38	5	33	21	50	262	3910	98	6501	20
70126	3.	10.	3	0	2	1	4	23	345	8	573	1
70127	1.	3.	1	0	0	0	1	7	115	2	191	0
70129	0.	0.	0	0	0	0	0	0	0	0	0	0
70130	0.	0.	0	0	0	0	0	0	0	0	0	0
70140	109.	347.	122	16	108	67	163	840	12536	315	20844	65
70150	0.	0.	0	0	0	0	0	0	0	0	0	0
70169	0.	0.	0	0	0	0	0	0	0	0	0	0
TOTAL	1301.	4135.	1449	182	1274	792	1929	10013	149628	3752	248780	770

Hospitals, and consequently surgical teams, are assumed by the model to be located in one grid; ZIP Code 70115 was chosen because the majority of the hospital facilities are located in this area The number of surgical teams in this grid was assumed to be one-sixth of the number of surviving physicians in the grid.

The format for these resource input data processed by the model is described in detail in Appendix D.

B. Results

The distribution of the 387,000 casualties considered during the Immediate Effects time period (60 days postattack) is given in Table XIII by type of preferred treatment and treatment actually given.

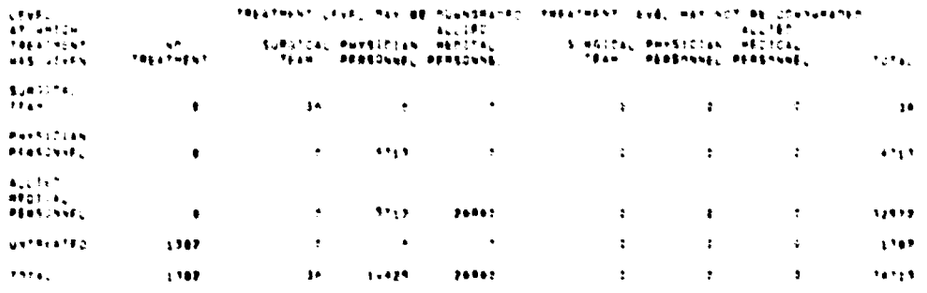
A total of 5436 medical personnel survived the attack; i.e., 1301 physicians and 4135 allied medical personnel. As stated previously, simulating the New Orleans postattack medical situation using the preferred treatment levels described in Appendix B resulted in no "downgrading" of treatment level; i.e., adequate personnel were available at the preferred treatment level for each injury. However, using upgraded treatment levels shown in Table X required more physician time than was available. This is illustrated in Table XIII, which shows that 57,120 of the 104,250 casualties for whom physician treatment was preferred were actually treated by allied medical personnel.

Further examination of Table XIII serves to indicate that a sufficient number of allied medical personnel survived because they were able to treat all casualties (325,720) assigned to them; i.e., the 268,600 who were designated as being in the allied medical personnel preferred treatment level, and the 57,120 who were downgraded from the physician level of preferred treatment. Note that the row labeled as "untreated" shows that only 13,820 casualties received no treatment. However, all of these casualties are listed under the column labeled "no treatment"; i.e., those casualties that were so severely injured that treatment would be of no avail, irrespective of its level, or those with minor injuries that require no medical attention to assure recovery.

Table XIII

NUMBER OF CASUALTIES AT EACH ASSIGNED LEVEL OF TREATMENT

(All Values are in Tens)



The medical treatment packages available initially for treatment of these casualties, the numbers used, and those remaining after the 60 day Immediate Effects period are shown in Table XIV. These totals for the entire City of New Orleans indicate that sufficient treatment packages were always available. However, a detailed analysis of results for each of the ZIP Code areas indicated that the supplies of several types of packages were depleted and the model does not consider movement of medical supplies or personnel from one grid to another. For example, the supply of Treatment Package BI, which is used in the treatment of severe burns, was inadequate in 20 of the 24 ZIP Code areas.

Table XIV

STATUS OF INVENTORY OF EACH MEDICAL TREATMENT PACKAGE
(All Values are in Tens)

Treatment Package	BI	A	T	P	S	M	AF	BA
Initial	146	18	128	79	196	1000	0	14945
Final	37	11	78	48	193	413	0	13575
Used	109	7	50	31	3	587	0	1370

Treatment Package	LF	W	H	HF	FF	E	V
Initial	0	376	24842	0	0	0	77
Final	0	107	24609	0	0	0	75
Used	0	269	233	0	0	0	2

The numbers of deaths during the Immediate Effects time period are reported by level of treatment in Table XV, which shows a total of 184,900 deaths; this is approximately 18.5 percent of the preattack population. A comparison of those deaths with the casualty data reported in Table XIII indicates that deaths occurred for 47.8 percent of the 387,030 casualties. The high death rate among the injured is due primarily to the high mortality (167,000) from fallout (it was assumed that medical attention did not affect the number of deaths-- is assumption could be in error). Deaths expressed as a percentage of casualties, by level of preferred treatment, were 40.8 percent for "No Treatment", 27.8 percent for "Surgical Teams", 10.8 percent for "Physicians", and 62.5 percent for "Allied Medical Personnel".

Table XV

NUMBER OF DEATHS AT EACH ASSIGNED TREATMENT LEVEL

(All Values are in Tens)

LEVEL OF MEDICAL TREATMENT WAS GIVEN	NO TREATMENT	TREATMENT LEVEL MAY BE DOWNGRADED			TREATMENT LEVEL MAY NOT BE DOWNGRADED			
		SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	TOTAL
SURGICAL TEAM	0	1*	*	*	0	0	0	1*
PHYSICIAN PERSONNEL	0	0	9**	0	0	0	0	9**
ALLIED MEDICAL PERSONNEL	*	*	1**	10***	0	*		10***
UNTREATED	***	0	*	0	0	0	0	***
TOTAL	***	1*	11**	10***	0	0	0	1*****

Upper and lower bounds for fatalities in the Immediate Effects Submodel were obtained by using the respective probabilities of death associated with "no treatment" and with "adequate treatment." These results indicate that 184,000 deaths would occur if everyone received medical care and 188,000 deaths would occur if no medical care was available.

III. DISEASE AND CHRONIC CONDITIONS SUBMODEL

A. Input Data

The 817,000 survivors of the Immediate Effects Submodel were used as input for the Disease and Chronic Conditions Submodel and subjected to nine diseases under the simulated postattack conditions of this study. The nine diseases are: Influenza, Paratyphoid B, Bacterial Dysentery, Infectious Hepatitis, Whooping Cough, Measles, Diphtheria, Gastroenteritis, and Scarlet Fever. Other communicable diseases are presently absent in most major U. S. Cities, and could be prevented by effective quarantine. Pneumococcal pneumonia is not considered in this test run, since it would act as a complicating disease, rather than as an epidemic, per se.

A brief discussion of the values assigned to the parameters used to analyze the effects of these diseases on the surviving population and the method by which they were calculated from preattack equilibrium conditions is presented below:

1) The fraction of the infected persons that will become infective themselves is called "infective ratio" (AL) and is, for the purpose of obtaining preattack equilibrium values, assumed to equal 0.5. Under postattack conditions, it is assumed to have increased to 0.75 due to radiation exposure.

2) The initial infective fraction (SSS) was obtained by assuming (a) an influx of new susceptibles into the population (mainly as newborn) equalling 2 percent per year (which is 2 per 36,500 person-days) and (b) a value 0.5 for the preattack infective ratio (AL). Hence, the initial infective fraction equals D/36,500; where D is the duration of infectivity in an infective person, as obtained from various textbooks.

3) The initial susceptible fraction (SR) was obtained from the time interval ($\tilde{t}$) between two consecutive wave peaks by applying the formula $s = \tilde{t}\,m$ (s = susceptible fraction; m = birth rate) to preattack data obtained from the literature (see Section III of Chapter 3.

4) The contact rate (TB) was calculated with the formula: TB = 1./(SR*AL*DIN), where AL = infective ratio and DIN is duration of the infectivity in an infective. Only for influenza could the contact rate be calculated more directly from the observations (see Section III of Chapter 3).

In addition to using intravenous infusion units and broad spectrum antibiotics (ampicillin equivalent) that were not consumed by the Immediate Effects Submodel, penicillin and sulfa were also required for the Disease and Chronic Conditions Submodel of the total model. The surviving penicillin and sulfa were calculated from the preattack total in a manner similar to that described in Section II. A.2. of this Chapter.

3. Results

Figure 11 provides a comprehensive picture of the postattack posture of New Orleans under the assumption that an unlimited supply of physicians is available. The number of diseased (infective) individuals and the number of physicians required for treatment are plotted for each 5-day interval throughout the entire "Disease and Chronic Condition Period" of 30-365 days.

Further attention should be given to two peaks that occur in both graphs--a sharp peak at 55 days and a broad peak around 150 days. The 55th-day peak can be attributed to a widespread epidemic of gastroenteritis, whereas the broad 150th-day peak can be blamed for the most part to a combination of three diseases; i.e., bacterial dysentery, paratyphoid, and influenza (see Table XVI). The shaded area of the "Physicians Required" curve represents physician time that exceeds the estimated time available.

Table XVII presents, by 5-day intervals, the drug requirements for the epidemics. It is apparent that the drug supply in New Orleans was inadequate with respect to intravenous infusion units (57,000 units available--290,104 units required), penicillin (281,000 M units available--895,806 M units required), and broad spectrum antibiotics (ampicillin equivalent) (971,000 grams available--1,430,804 grams required). On the other hand, there was a sufficient quantity of sulfa available.

It is important to note that under these ideal "drug and physician" conditions only 15,494 or 1.9 percent of the original 817,000 survivors of the Immediate Effects period died from infectious diseases during the ensuing year (see Table XVIII).

In order to provide a more accurate picture of the "postattack disease problem" in New Orleans, a simulation run was made utilizing those medical resources estimated to actually be available (see Section III.A. of this Chapter). This more realistic approach resulted in 31,485 deaths as shown in Table XIX. This represents 3.85 percent of the 817,000 survivors of the Immediate Effects time period and is 15,991 more fatalities than would occur with maximum medical resources available. A comparison of disease death totals in Tables XVIII and XIX indicates that most of the added fatalities (11,653) can be attributed to gastroenteritis. This fact is easily explained by the short supply of intravenous infusion units that are needed to treat this disease.

An upper limit to this problem was obtained by simulating the New Orleans study with no drugs and no physicians available (see Table XX). These results show that 284,835 fatalities would occur under these conditions--35 percent of the survivors of the Immediate Effects phase or an 1,839 percent increase over the minimum estimate (maximum drugs and physicians available) and a 905 percent increase over the "realistic" estimates.

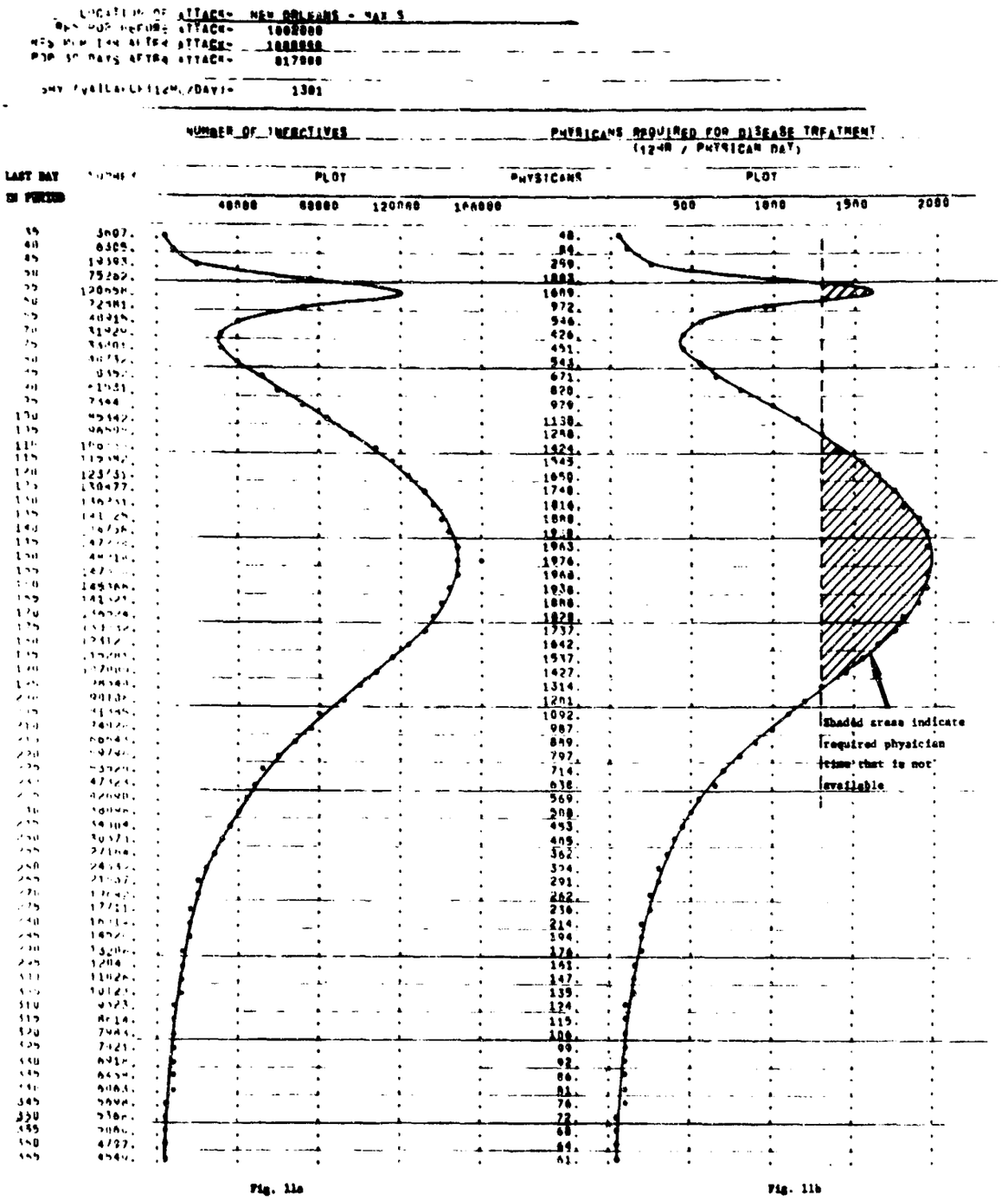


Fig. 11. Plot of Infectives and Required Physicians.

Table XVI

NUMBER OF NEW INFECTIVES
BY DISEASE AND BY 5-DAY PERIOD

LOCATION OF ATTACK= NEW ORLEANS - MAY 5
DEN FOR BEFORE ATTACK= 1002000
DEN FOR IMM AFTER ATTACK= 1000000
DAYS AFTER ATTACK= 817000

DAY IN PERIOD	DISEASE									
	[illegible]	MISE	GAST	MEPA	INFL	MEAS	PARA	SCAR	WHOO	TOTAL
[illegible]	[illegible]	180.	[illegible]	180.	172.	139.	180.	180.	155.	1749.
40	[illegible]	229.	2517.	245.	270.	204.	212.	262.	163.	4344.
45	[illegible]	311.	17243.	360.	417.	294.	262.	384.	172.	19833.
50	[illegible]	417.	49457.	515.	621.	425.	335.	464.	180.	93174.
55	[illegible]	556.	108421.	719.	915.	589.	417.	409.	188.	185829.
60	[illegible]	744.	118218.	989.	1324.	793.	523.	1152.	204.	125120.
65	[illegible]	967.	48140.	1340.	1904.	1045.	662.	1618.	221.	57594.
70	[illegible]	1242.	17476.	1757.	2705.	1314.	825.	2223.	237.	30375.
75	[illegible]	1765.	6301.	2242.	3800.	1594.	1030.	2975.	253.	23077.
[illegible]	[illegible]	2367.	2198.	2754.	5263.	1822.	1241.	3857.	270.	23870.
85	[illegible]	3041.	735.	3228.	7175.	1978.	1410.	4789.	294.	28022.
90	[illegible]	4047.	212.	3598.	9561.	2035.	2002.	5647.	311.	33538.
95	[illegible]	5245.	31.	3800.	12397.	1986.	2484.	6301	327.	39519.
100	[illegible]	6788.	0.	3824.	15527.	1847.	3073.	6519.	345.	45387.
105	[illegible]	8605.	0.	3661.	18640.	1659.	3775.	6578.	368.	50707.
110	[illegible]	10771.	0.	3375.	21321.	1438.	4417.	6219.	384.	53193.
115	[illegible]	13222.	0.	3007.	23135.	1224.	5014.	5630.	409.	53681.
120	[illegible]	15845.	0.	2607.	24280.	1021.	6766.	4928.	425.	61034.
125	[illegible]	18469.	0.	2215.	24208.	843.	8068.	4200.	449.	62288.
130	[illegible]	20885.	0.	1855.	23598.	686.	9520.	3506.	466.	62524.
135	[illegible]	22753.	0.	1536.	22286.	556.	11073.	2985.	482.	61927.
140	[illegible]	24014.	0.	1258.	20446.	443.	12491.	2545.	507.	60836.
145	[illegible]	24845.	0.	1021.	18974.	369.	14285.	1896.	523.	58721.
150	[illegible]	24140.	0.	834.	31482.	294.	15788.	1520.	539.	56337.
155	[illegible]	23110.	0.	670.	3275.	245.	17096.	1218.	556.	53559.
160	[illegible]	21588.	0.	548.	7404.	195.	18117.	972.	572.	50470.
165	[illegible]	19878.	0.	441.	5851.	161.	18795.	769.	588.	47160.
170	[illegible]	17843.	0.	360.	4584.	139.	19093.	615.	597.	43729.
175	[illegible]	15992.	0.	294.	3579.	114.	19016.	490.	613.	40247.
180	[illegible]	13815.	0.	237.	2787.	94.	18575.	384.	621.	36749.
185	[illegible]	11778.	0.	196.	2157.	82.	17448.	311.	629.	33341.
190	[illegible]	10109.	0.	163.	1675.	74.	16483.	245.	637.	30056.
195	[illegible]	8621.	0.	131.	1299.	65.	15764.	196.	637.	26927.
200	[illegible]	7322.	0.	114.	1005.	57.	14554.	155.	646.	24029.
205	[illegible]	6194.	0.	90.	785.	49.	13304.	131.	646.	21337.
210	[illegible]	5222.	0.	82.	613.	49.	12262.	106.	646.	18993.
215	[illegible]	4348.	0.	65.	482.	49.	10852.	82.	646.	16654.
220	[illegible]	3648.	0.	57.	378.	41.	9716.	65.	637.	14652.
225	[illegible]	3049.	0.	57.	302.	41.	8454.	57.	637.	12895.
230	[illegible]	2582.	0.	49.	245.	41.	7473.	49.	629.	11518.
235	[illegible]	2187.	0.	41.	196.	41.	6783.	41.	621.	9921.
240	[illegible]	1798.	0.	41.	163.	41.	5974.	33.	613.	8695.
245	[illegible]	1469.	0.	41.	131.	41.	5255.	33.	605.	7633.
250	[illegible]	1240.	0.	33.	114.	41.	4609.	25.	597.	6693.
255	[illegible]	1048.	0.	33.	98.	33.	4037.	25.	588.	5879.
260	[illegible]	868.	0.	33.	90.	33.	3522.	25.	572.	5168.
265	[illegible]	719.	0.	33.	82.	33.	3081.	16.	564.	4544.
270	[illegible]	597.	0.	33.	74.	33.	2680.	16.	548.	3994.
275	[illegible]	498.	0.	33.	65.	33.	2337.	16.	539.	3538.
280	[illegible]	418.	0.	25.	65.	33.	2035.	16.	523.	3122.
285	[illegible]	343.	0.	25.	57.	33.	1773.	16.	507.	2770.
290	[illegible]	278.	0.	25.	57.	41.	1536.	16.	498.	2460.
295	[illegible]	229.	0.	25.	57.	41.	1340.	16.	482.	2190.
300	[illegible]	188.	0.	25.	49.	41.	1160.	16.	466.	1953.
305	[illegible]	155.	0.	25.	49.	41.	1005.	16.	449.	1749.
310	[illegible]	131.	0.	25.	49.	41.	874.	16.	441.	1586.
315	[illegible]	106.	0.	25.	49.	41.	760.	16.	425.	1430.
320	[illegible]	82.	0.	25.	49.	41.	654.	16.	409.	1283.
325	[illegible]	65.	0.	25.	49.	41.	572.	16.	400.	1177.
330	[illegible]	57.	0.	25.	49.	41.	490.	16.	384.	1071.
335	[illegible]	41.	0.	25.	49.	41.	425.	16.	376.	981.
340	[illegible]	33.	0.	25.	49.	41.	368.	16.	360.	899.
345	[illegible]	25.	0.	25.	49.	41.	319.	16.	151.	834.
350	[illegible]	16.	0.	25.	49.	41.	278.	16.	335.	768.
355	[illegible]	16.	0.	25.	49.	41.	237.	16.	327.	719.
360	[illegible]	8.	0.	25.	49.	41.	204.	16.	311.	662.
365	[illegible]	8.	0.	33.	49.	41.	180.	16.	302.	637.
TOTAL	92491.	397995.	484040.	51241.	299455.	27153.	387577.	83448.	30398.	1861757.

Table XVII

DRUG REQUIREMENTS - BY 5-DAY PERIOD AND CUMULATIVE TOTAL

LOCATION OF ATTACK- NEW ORLEANS - MAX 5	
RES POP BEFORE ATTACK-	1002000
RES POP IMM AFTER ATTACK-	817000
POP 30 DAYS AFTER ATTACK-	1000000
INT INFUSION AVAILABLE -	57000 UNITS
PENICILLIN AVAILABLE -	281000 M UNITS
SULFA AVAILABLE -	1268000 GRAMS
B-S ANTIBIO (AMPICILLIN EQUIVALENT) AVAILABLE -	971000 GRAMS

LAST DAY IN PERIOD	INTRAVENOUS INFUSION UNITS REQUIRED	TOTAL REQUIRED	PENICILLIN MILLIONS OF UNITS REQUIRED	TOTAL REQUIRED	SULFA GRAMS REQUIRED	TOTAL REQUIRED	AMPICILLIN GRAMS REQUIRED	TOTAL REQUIRED
35	288.	288.	1357.	1357.	385.	385.	451.	451.
40	2011.	2298.	1934.	3291.	503.	888.	542.	992.
45	13774.	16073.	2701.	5992.	691.	1579.	690.	1688.
50	47838. →	63911.	3798.	9790.	918.	2497.	881.	2569.
55	110720.	174631.	5312.	15102.	1233.	3730.	1101.	3670.
60	59559.	234190.	7388.	22490.	1650.	5379.	1385.	5056.
65	23149.	257339.	10182.	32672.	2204.	7584.	1743.	6799.
70	8391.	265729.	13854.	46526.	2941.	10525.	2185.	8984.
75	2963.	268692.	18534.	65060.	3915.	14440.	2741.	11724.
80	1016.	269708.	24261.	89321.	5194.	19638.	3437.	15161.
85	322.	290030.	30915.	120236.	6874.	26513.	4287.	19448.
90	75.	290104.	38158.	158394.	9047.	35560.	5342.	24790.
95	0.	290104.	45414.	203808.	11831.	47391.	6658.	31448.
100	0.	290104.	51939.	255747.	15344.	62736.	8263.	39711.
105	0.	290104.	56994. →	312741.	19760.	82496.	10718.	50429.
110	0.	290104.	59997.	372738.	24899.	107395.	12080.	62509.
115	0.	290104.	60635.	433373.	31035.	138393.	15406.	77915.
120	0.	290104.	58906.	492278.	37864.	176257.	18744.	96659.
125	0.	290104.	55126.	547404.	45087.	221344.	22627.	119286.
130	0.	290104.	49851.	597254.	52215.	273559.	27060.	146346.
135	0.	290104.	43788.	641042.	58587.	332146.	32007.	178353.
140	0.	290104.	37446.	678488.	63689.	395835.	37379.	215731.
145	0.	290104.	31420.	709908.	66875.	462710.	43017.	258748.
150	0.	290104.	25935.	735842.	67934.	530644.	48703.	307451.
155	0.	290104.	21253.	757094.	66835.	597480.	54159.	361610.
160	0.	290104.	17282.	774376.	63934.	661414.	59069.	420679.
165	0.	290104.	14019.	788394.	59680.	721094.	63139.	483818.
170	0.	290104.	11383.	799776.	54352.	775446.	66105.	549924.
175	0.	290104.	9281.	809057.	48810.	824256.	67800.	617724.
180	0.	290104.	7622.	816678.	43144.	867400.	68161.	685884.
185	0.	290104.	6322.	823000.	37648.	905048.	67238.	753122.
190	0.	290104.	5309.	828308.	32560.	937608.	65177.	818298.
195	0.	290104.	4523.	832830.	28092.	965700.	62185.	880482.
200	0.	290104.	3914.	836742.	23896.	989596.	58501.	938982.
205	0.	290104.	3442.	840184.	20294.	1009890.	54363. →	993344.
210	0.	290104.	3079.	843262.	17160.	1027050.	49983.	1043328.
215	0.	290104.	2796.	846058.	14468.	1041518.	45536.	1088864.
220	0.	290104.	2578.	848636.	12172.	1053690.	41172.	1130036.
225	0.	290104.	2408.	851044.	10194.	1063884.	36980.	1167016.
230	0.	290104.	2272.	853316.	8544.	1072428.	33032.	1200048.
235	0.	290104.	2166.	855482.	7144.	1079572.	29368.	1229416.
240	0.	290104.	2058.	857540.	5964.	1085536.	26008.	1255424.
245	0.	290104.	2006.	859546.	4976.	1090512.	22956.	1278380.
250	0.	290104.	1966.	861512.	4144.	1094656.	20204.	1298584.
255	0.	290104.	1892.	863404.	3452.	1098108.	17744.	1316328.
260	0.	290104.	1846.	865250.	2868.	1100976.	15548.	1331876.
265	0.	290104.	1804.	867054.	2384.	1103360.	13600.	1345476.
270	0.	290104.	1765.	868818.	1980.	1105340.	11882.	1357358.
275	0.	290104.	1726.	870544.	1608.	1106948.	10412.	1367770.
280	0.	290104.	1690.	872234.	1392.	1108340.	9022.	1376792.
285	0.	290104.	1654.	873888.	1104.	1109444.	7824.	1384616.
290	0.	290104.	1620.	875508.	929.	1110302.	6840.	1391456.
295	0.	290104.	1589.	877096.	765.	1111146.	5944.	1397400.
300	0.	290104.	1552.	878648.	638.	1111784.	5164.	1402564.
305	0.	290104.	1508.	880156.	512.	1112296.	4880.	1407444.
310	0.	290104.	1482.	881638.	420.	1112716.	3988.	1411432.
315	0.	290104.	1448.	883086.	342.	1113058.	3368.	1414800.
320	0.	290104.	1414.	884500.	274.	1113332.	2420.	1417220.
325	0.	290104.	1382.	885882.	221.	1113553.	2550.	1419770.
330	0.	290104.	1350.	887232.	171.	1113724.	2166.	1421936.
335	0.	290104.	1316.	888548.	137.	1113861.	1861.	1423797.
340	0.	290104.	1288.	889836.	105.	1113966.	1643.	1425440.
345	0.	290104.	1252.	891088.	77.	1114043.	1429.	1426869.
350	0.	290104.	1224.	892312.	57.	1114100.	1215.	1428084.
355	0.	290104.	1194.	893506.	32.	1114132.	1048.	1429132.
360	0.	290104.	1164.	894670.	21.	1114152.	900.	1430032.
365	0.	290104.	1136.	895806.	16.	1114168.	772.	1430804.

Note: Arrows indicate points at which available medical supplies are depleted.

Table XVIII

NUMBER OF DEATHS WITH MAXIMUM MEDICAL RESOURCES BY DISEASE AND 5-DAY PERIOD

LOCATION OF ATTACK- NEW ORLEANS - MAX 5
MEN POP BEFORE ATTACK- 1002000
MEN POP 1MO AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 857000

LAST DAY IN PERIOD	DIPH	DISE	GAST	HEPA	DISEASE INFL	MEAS	PARA	SCAR	WHOO	TOTAL
35	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
40	0.	0.	25.	0.	0.	0.	0.	0.	0.	25.
45	0.	0.	188.	0.	0.	0.	0.	0.	0.	196.
50	0.	0.	997.	0.	0.	0.	0.	0.	0.	1021.
55	0.	0.	2010.	0.	0.	0.	0.	0.	0.	2035.
60	10.	0.	1310.	0.	0.	0.	0.	0.	0.	1340.
65	10.	0.	531.	0.	10.	0.	0.	0.	0.	588.
70	29.	10.	190.	0.	10.	0.	0.	0.	0.	270.
75	55.	10.	74.	0.	25.	0.	0.	0.	0.	163.
80	41.	25.	25.	0.	41.	0.	0.	0.	0.	155.
85	57.	13.	0.	0.	49.	0.	0.	0.	0.	172.
90	59.	11.	0.	0.	65.	0.	16.	0.	0.	204.
95	74.	10.	0.	10.	90.	0.	10.	0.	0.	253.
100	82.	10.	0.	10.	114.	0.	25.	0.	0.	311.
105	82.	10.	0.	0.	131.	0.	25.	0.	0.	335.
110	82.	110.	0.	0.	155.	0.	33.	0.	0.	384.
115	71.	141.	0.	0.	163.	0.	41.	0.	0.	417.
120	69.	155.	0.	0.	172.	0.	49.	0.	0.	449.
125	57.	148.	0.	0.	165.	0.	57.	0.	0.	474.
130	41.	212.	0.	0.	155.	0.	65.	0.	0.	482.
135	33.	229.	0.	0.	139.	0.	74.	0.	0.	482.
140	33.	247.	0.	0.	123.	0.	82.	0.	0.	482.
145	25.	215.	0.	0.	98.	0.	98.	0.	0.	466.
150	16.	215.	0.	0.	82.	0.	106.	0.	0.	440.
155	10.	230.	0.	0.	65.	0.	114.	0.	0.	425.
160	0.	212.	0.	0.	49.	0.	123.	0.	0.	400.
165	0.	198.	0.	0.	41.	0.	123.	0.	0.	378.
170	0.	180.	0.	0.	33.	0.	131.	0.	0.	360.
175	0.	165.	0.	0.	25.	0.	131.	0.	0.	327.
180	0.	139.	0.	0.	16.	0.	123.	0.	0.	294.
185	0.	114.	0.	0.	16.	0.	123.	0.	0.	262.
190	0.	98.	0.	0.	0.	0.	114.	0.	0.	229.
195	0.	90.	0.	0.	0.	0.	106.	0.	0.	212.
200	0.	74.	0.	0.	0.	0.	98.	0.	0.	188.
205	0.	65.	0.	0.	0.	0.	90.	0.	0.	172.
210	0.	49.	0.	0.	0.	0.	82.	0.	0.	147.
215	0.	41.	0.	0.	0.	0.	74.	0.	0.	123.
220	0.	41.	0.	0.	0.	0.	65.	0.	0.	114.
225	0.	33.	0.	0.	0.	0.	57.	0.	0.	98.
230	0.	25.	0.	0.	0.	0.	49.	0.	0.	82.
235	0.	25.	0.	0.	0.	0.	49.	0.	0.	82.
240	0.	16.	0.	0.	0.	0.	41.	0.	0.	65.
245	0.	16.	0.	0.	0.	0.	33.	0.	0.	57.
250	0.	16.	0.	0.	0.	0.	33.	0.	0.	57.
255	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
260	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
265	0.	0.	0.	0.	0.	0.	25.	0.	0.	33.
270	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
275	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
280	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
285	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
290	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
295	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
300	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
305	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
310	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
315	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
320	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
325	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
330	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
335	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
340	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
345	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
350	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
355	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
360	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
365	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
TOTAL	987.	3980.	9377.	147.	2188.	41.	2878.	0.	172.	19694.

Table XIX

NUMBER OF DEATHS WITH ESTIMATED MEDICAL RESOURCES BY DISEASE AND BY 5-DAY PERIOD

LOCATION OF ATTACK- NEW ORLEANS - EST M
EST POP BEFORE ATTACK- 1000000
MES POP 1HR AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 817000

LAST DAY IN PERIOD	DISEASE DTPH	DTSE	GAST	HEPA	INFL	MEAS	PARA	SCAR	WHOO	TOTAL
15	0.	0.	14.	0.	0.	0.	0.	0.	0.	25.
20	0.	0.	90.	0.	0.	0.	0.	0.	0.	98.
25	0.	0.	421.	0.	0.	0.	0.	0.	0.	537.
30	16.	0.	3212.	0.	0.	0.	0.	0.	0.	3244.
35	25.	0.	6429.	0.	0.	0.	0.	0.	0.	6472.
40	41.	0.	4127.	0.	16.	0.	0.	0.	0.	4200.
45	49.	0.	1643.	0.	25.	0.	0.	0.	0.	1749.
50	82.	16.	582.	0.	33.	0.	0.	0.	0.	727.
55	90.	16.	284.	0.	49.	0.	0.	0.	0.	384.
60	114.	25.	74.	0.	65.	16.	0.	0.	0.	311.
65	147.	41.	25.	16.	90.	16.	16.	0.	0.	351.
70	172.	41.	0.	16.	114.	16.	16.	0.	0.	392.
75	196.	49.	0.	16.	147.	16.	25.	0.	0.	458.
80	204.	65.	0.	16.	188.	16.	25.	0.	0.	531.
85	204.	90.	0.	16.	221.	16.	33.	0.	0.	587.
90	196.	106.	0.	16.	253.	0.	41.	0.	0.	537.
95	180.	131.	0.	0.	278.	0.	49.	0.	0.	470.
100	155.	155.	0.	0.	286.	0.	57.	0.	0.	486.
105	131.	188.	0.	0.	278.	0.	74.	0.	0.	705.
110	114.	212.	0.	0.	262.	0.	82.	0.	0.	695.
115	90.	229.	0.	0.	229.	0.	98.	0.	0.	670.
120	74.	247.	0.	0.	196.	0.	114.	0.	0.	537.
125	57.	245.	0.	0.	143.	0.	131.	0.	0.	413.
130	49.	245.	0.	0.	119.	0.	139.	0.	0.	480.
135	41.	229.	0.	0.	114.	0.	155.	0.	0.	548.
140	33.	212.	0.	0.	90.	0.	163.	0.	0.	507.
145	25.	196.	0.	0.	74.	0.	163.	0.	0.	466.
150	16.	180.	0.	0.	57.	0.	122.	0.	0.	433.
155	16.	155.	0.	0.	41.	0.	172.	0.	0.	392.
160	0.	139.	0.	0.	33.	0.	163.	0.	0.	351.
165	0.	114.	0.	0.	25.	0.	155.	0.	0.	311.
170	0.	98.	0.	0.	16.	0.	147.	0.	0.	278.
175	0.	90.	0.	0.	16.	0.	139.	0.	0.	262.
180	0.	74.	0.	0.	0.	0.	131.	0.	0.	229.
185	0.	65.	0.	0.	0.	0.	114.	0.	0.	196.
190	0.	49.	0.	0.	0.	0.	106.	0.	0.	172.
195	0.	41.	0.	0.	0.	0.	98.	0.	0.	155.
200	0.	41.	0.	0.	0.	0.	98.	0.	0.	147.
205	0.	33.	0.	0.	0.	0.	74.	0.	0.	114.
210	0.	25.	0.	0.	0.	0.	65.	0.	0.	98.
215	0.	25.	0.	0.	0.	0.	57.	0.	0.	90.
220	0.	16.	0.	0.	0.	0.	49.	0.	0.	74.
225	0.	16.	0.	0.	0.	0.	49.	0.	0.	74.
230	0.	16.	0.	0.	0.	0.	41.	0.	0.	65.
235	0.	0.	0.	0.	0.	0.	33.	0.	0.	49.
240	0.	0.	0.	0.	0.	0.	33.	0.	0.	49.
245	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
250	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
255	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
260	0.	0.	0.	0.	0.	0.	16.	0.	0.	33.
265	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
270	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
275	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
280	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
285	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
290	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
295	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
300	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
305	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
310	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
315	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
320	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
325	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
330	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
335	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
340	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
345	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
350	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
355	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
360	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
365	0.	0.	0.	0.	0.	0.	8.	0.	0.	16.
TOTAL	2330.	3080.	17030.	180.	3363.	180.	3490.	74.	400.	31485.

Table XX

NUMBER OF DEATHS WITH NO MEDICAL RESOURCES BY DISEASE AND BY 5-DAY PERIOD

LOCATION OF ATTACK- NEW ORLEANS - MIN R
RES POP BEFORE ATTACK- 1002000
RES POP IMM AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 817000

LAST DAY IN PERIOD	DIPH	DYSE	GAST	HEPA	DISEASE INFL	MEAS	PARA	SCAR	WHOO	TOTAL
35	41.	65.	123.	0.	8.	3.	8.	0.	9.	262.
40	57.	82.	817.	0.	8.	5.	8.	0.	8.	989.
45	90.	115.	7771.	0.	16.	16.	16.	8.	8.	8082.
50	131.	147.	27620.	8.	25.	25.	16.	0.	8.	27980.
55	150.	204.	49481.	8.	41.	33.	16.	8.	8.	49971.
60	202.	270.	26322.	8.	57.	41.	25.	0.	8.	26992.
65	366.	360.	8286.	16.	74.	57.	33.	8.	8.	9210.
70	507.	474.	2313.	16.	106.	65.	41.	8.	16.	3547.
75	530.	629.	824.	25.	155.	82.	49.	8.	16.	2272.
80	493.	814.	158.	33.	212.	98.	65.	8.	16.	2304.
85	1072.	1103.	33.	33.	288.	106.	82.	16.	16.	2770.
90	1221.	1448.	0.	41.	384.	106.	98.	16.	16.	3301.
95	1433.	1863.	0.	41.	498.	98.	123.	16.	16.	4086.
100	1487.	2378.	0.	41.	621.	90.	147.	16.	16.	4797.
105	1405.	3007.	0.	41.	744.	82.	180.	16.	16.	5540.
110	1357.	3718.	0.	33.	850.	74.	221.	16.	25.	6292.
115	1291.	4511.	0.	33.	915.	57.	270.	16.	25.	7028.
120	1031.	5320.	0.	25.	940.	49.	327.	16.	25.	7731.
125	850.	6056.	0.	25.	915.	41.	392.	8.	25.	8352.
130	693.	6742.	0.	16.	850.	33.	458.	8.	25.	8826.
135	550.	7193.	0.	16.	752.	25.	531.	8.	25.	9095.
140	433.	7379.	0.	16.	646.	25.	613.	8.	25.	9144.
145	343.	7306.	0.	8.	539.	16.	686.	8.	33.	8940.
150	271.	6947.	0.	8.	441.	16.	760.	8.	33.	8523.
155	201.	6440.	0.	8.	351.	8.	817.	0.	33.	7989.
160	163.	5851.	0.	8.	288.	8.	866.	0.	33.	7218.
165	123.	5165.	0.	8.	221.	8.	899.	0.	33.	6458.
170	98.	4470.	0.	0.	172.	8.	907.	0.	33.	5689.
175	74.	3818.	0.	0.	139.	8.	899.	0.	33.	4960.
180	57.	3220.	0.	0.	106.	8.	874.	0.	33.	4298.
185	41.	2659.	0.	0.	82.	8.	842.	0.	33.	3666.
190	33.	2231.	0.	0.	65.	8.	793.	0.	33.	3154.
195	25.	1819.	0.	0.	49.	0.	735.	0.	33.	2660.
200	25.	1504.	0.	0.	41.	0.	678.	0.	33.	2280.
205	16.	1229.	0.	0.	33.	0.	621.	0.	33.	1920.
210	16.	967.	0.	0.	25.	0.	556.	0.	33.	1626.
215	8.	[illegible]	0.	0.	16.	0.	498.	0.	33.	1368.
220	8.	[illegible]	0.	0.	16.	0.	449.	0.	33.	1169.
225	8.	541.	0.	0.	8.	0.	392.	0.	33.	[illegible]
230	8.	413.	0.	0.	8.	3.	351.	0.	33.	814.
235	8.	361.	0.	0.	8.	0.	311.	0.	33.	711.
240	8.	274.	0.	0.	8.	0.	278.	0.	33.	607.
245	8.	259.	0.	0.	8.	0.	237.	0.	33.	547.
250	8.	[illegible]	0.	0.	8.	0.	204.	0.	33.	429.
255	8.	147.	0.	0.	0.	0.	180.	0.	33.	368.
260	8.	114.	0.	0.	0.	0.	155.	0.	33.	[illegible]
265	8.	90.	0.	0.	0.	0.	139.	0.	33.	262.
270	8.	74.	0.	0.	0.	0.	123.	0.	33.	229.
275	0.	57.	0.	0.	0.	0.	106.	0.	33.	196.
280	0.	49.	0.	0.	0.	0.	77.	0.	25.	163.
285	0.	41.	0.	0.	0.	0.	74.	0.	25.	139.
290	0.	33.	0.	0.	0.	0.	65.	0.	25.	123.
295	0.	25.	0.	0.	0.	0.	57.	0.	25.	106.
300	0.	16.	0.	0.	0.	0.	49.	0.	25.	90.
305	0.	16.	0.	0.	0.	0.	41.	0.	25.	82.
310	0.	8.	0.	0.	0.	0.	41.	0.	25.	74.
315	0.	8.	0.	0.	0.	0.	33.	0.	25.	65.
320	0.	8.	0.	0.	0.	0.	25.	0.	25.	57.
325	0.	8.	0.	0.	0.	0.	25.	0.	25.	57.
330	0.	8.	0.	0.	0.	0.	25.	0.	16.	49.
335	0.	0.	0.	0.	0.	0.	16.	0.	16.	33.
340	0.	0.	0.	0.	0.	0.	16.	0.	16.	33.
345	0.	0.	0.	0.	0.	0.	16.	0.	16.	33.
350	0.	0.	0.	0.	0.	0.	8.	0.	16.	25.
355	0.	0.	0.	0.	0.	0.	8.	0.	16.	25.
360	0.	0.	0.	0.	0.	0.	8.	0.	16.	25.
365	0.	0.	0.	0.	0.	0.	8.	0.	16.	25.
TOTAL	17686.	111861.	121330.	313.	11735.	1299.	18672.	212.	1630.	304035.

IV. SUMMARY AND CONCLUSIONS

A. Summary

The Total Emergency Health Care System Model was used to examine the postattack health posture of New Orleans, Louisiana following a surface burst of a 1.5 MT thermonuclear weapon at a point approximately 9 miles south of the city's center. The Model simulates a one-year postattack period employing an Immediate Effects Submodel (operating to 60 days postattack) and a Disease and Chronic Conditions Submodel (operating out to one year). Casualties from direct weapons effects serve as input to the Immediate Effects Submodel; surviving injured and the uninjured population are input to the Disease Submodel and analyzed as to their ability to survive nine communicable diseases under one year postattack conditions.

Latest Dikewood casualty curves for this weapon were applied to each Zip Code area to obtain the number and type of expected injuries. Medical supply data were furnished by the Public Health Service, numbers of physicians were supplied by OCD, and fallout casualties were obtained from a READY run. Surviving medical supplies were estimated to be proportionate to the surviving physicians.

Approximately 387,000 or 39 percent of the 1,002,000 preattack inhabitants of New Orleans were injured by the weapon, either through fallout and/or direct effects--and 185,000 or 47.8 percent of these injuries were fatal. Most of these fatalities (173,000 or 93.5 percent of direct effects fatalities) were due directly or indirectly to the effects of fallout radiation.

Included among these survivors were 5,436 medical personnel; i.e., 1,301 physicians and 4,135 allied medical personnel; surviving physicians proved to be inadequate during both the Imeediate Effects and the one year Communicable Diseases and Chronic Conditions postattack periods.

Of the 817,000 people who survived the immediate effects time period, approximately 31,485 or 3.85 percent died from infectious diseases during the ensuing year.

It was found that New Orleans had an adequate overall supply of the various medical treatment packages required for treating casualties during the immediate effects period although shortages of some types of packages (especially those needed for severe burns) were noted in several of the 24 Zip Code areas. However, as shown in Table XXI, three of the four drug supplies required for the one year postattack Communicable Disease and Chronic Conditions Submodel period were inadequate. These shortages were contributing factors to the high number of fatalities (31,485) attributed to infectious diseases.

Table XXI

DRUGS REQUIRED AND AVAILABLE FOR COMMUNICABLE DISEASE PHASE
(New Orleans Case Study)

	Required	Available (On Hand)
Intravenous Infusion (Units)	290,104	57,000
Penicillin (Millions of Units)	895,806	281,000
Sulfa (Grams)	1,114,160	1,268,000
Broad-Spectrum Antibiotics (Grams) (Ampicillin Equivalent)	1,430,804	971,000

In order to provide more insight into the results of this study, upper and lower bounds of fatalities were estimated by applying death probabilities based on the availability of unlimited medical resources (lower bound) and no medical resources (upper bound). The results of these computation are presented in Table XXII and in Fig. 12.

Table XXII

INJURIES AND FATALITIES FOR THREE LEVELS OF MEDICAL SUPPLIES
(New Orleans Case Study)

	Deaths			Injuries
Cause of Death	Unlimited Medical Resources	Estimated Available Resources	No Medical Resources	
Instantaneous Direct Effects	2,000	2,000	2,000	-
Immediate Effects (60 Days)				
a) Fallout Only	167,000	167,000	167,000	282,000
b) Injuries (No Fallout)	9,400	10,000	12,550	91,500
c) Injuries (Complicated by Fallout)	5,600	6,000	6,450	13,500
Communicable Disease (30-365 Days)	15,494	31,485	284,835	-
Total	199,494	216,485	472,835	387,000
Percent Deaths of 1,002,000 Preattack Population	20%	22%	47%	

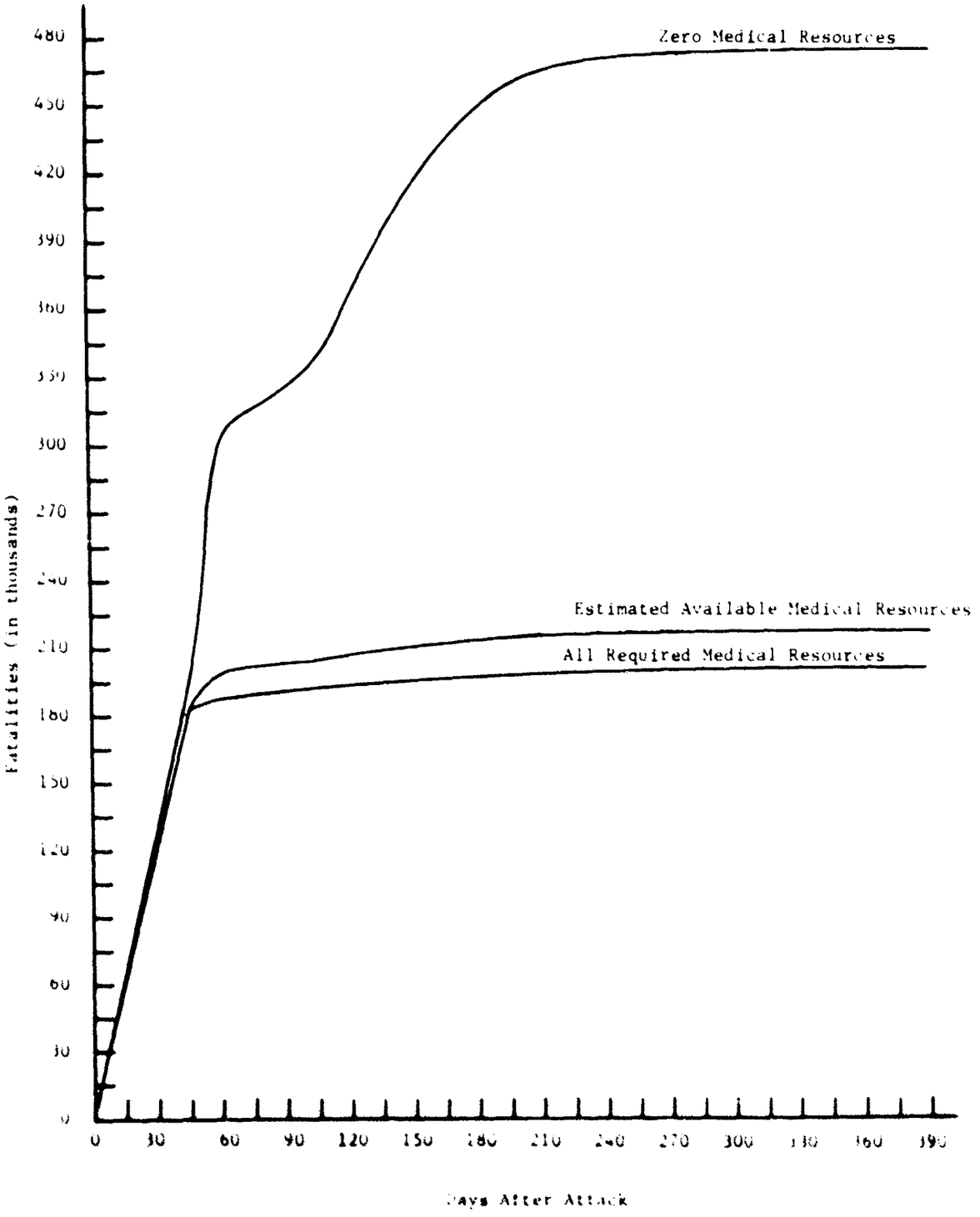


Fig. 12. Cumulative Deaths for New Orleans

It is important to note that although medical resources have relatively little effect on reducing fatalities during the 60 day Immediate Effects period, they do have a profound effect on the number of fatalities that occur during the ensuing year.

B. Conclusions

After taking into consideration those limitations imposed by data, assumptions, and parameters, the results of this study suggest the following conclusions.

1) Improvement in the emergency capabilities of New Orleans pertinent to the 60-day Immediate Effects Postattack Period would result in a small reduction of fatalities (185,000 to 184,000). This is due primarily to the fact that fallout radiation was a predominant factor in the number of fatal and non-fatal injuries suffered during this period. A slight shortage of physicians was also indicated but an adequate number of medical treatment packages and surviving allied medical personnel served to minimize this discrepancy.

2) A shortage of required drugs was evident during the one-year Communicable Disease and Chronic Conditions phase. Since projected upper and lower fatality limits for this period indicate that preventable deaths are highly dependent upon these supplies, preattack medical supply planning and stockpiling of required drugs are suggested--especially with respect to Intravenous Infusion and penicillin.

3) A lack of available physicians, though apparent throughout the entire postattack period, was especially criti during the epidemics that occurred around the 55th and 150th days of the Communicable Disease and Chronic Conditions time period.

BIBLOGRAPHY

Abbey, H. "An Examination of the Reed-Frost Theory of Epidemics." Human Biology, Vol. 24, 1952, pp. 201-233.

Acherknecht, Erwin H. History and Geography of the Most Important Diseases. New York: Hafner Publishing Company, 1965.

Andrewes, C. H. "Adventures Among Viruses. III. The Puzzel of the Common Cold," New England Journal of Medicine. Vol. 242, 1950, pp. 235-240.

Bailey, N. T. J. The Mathematical Theory of Epidemics. New York: Hafner Publishing Company, 1957.

Bang, F. B. "Mucociliary Function as Protective Mechanism in Upper Respiratory Tract," Bacteriological Reviews. Vol. 25, 1961, pp. 228-236.

Bartlett, M. S. "Measles Periodicity and Community Size." Journal of Royal Statistical Society. Series A, Vol. 120, 1957, pp. 48-60.

Bartlett, M. S. Stochastic Processes. Cambridge: Cambridge University Press (1955).

"B. C. G. and Vole Bacillus Vaccines in the Prevention of Tuberculosis in Adolescents. First Report to the Council," British Medical Journal. No. 4964, Vol. 1, 1956, pp. 413-427.

Bell, J. A., et al. "Epidemiological Observations on Two Outbreaks of Asian Influenza in a Children's Institution," American Journal of Hygiene, Vol. 73, 1961, pp. 84-89.

Blair, J. E. "The Staphylococci " Bacterial and Mycotic Infections of Man (3rd edition), (ed. R. J. Dubos). London: Pitman, 1958, p. 315.

Borgen, L., S. N. Meyer, and E. Refsum. "Mass Photoflurography, Tuberculin Testing and B. C. G. Vaccination in the District of Akar (Norway) 1947-1949," Acta Tuberculosis Scandinavica, Vol. 25 (1950), pp. 327-355.

Brimblecombe, F. S. W., et al. "Family Studies of Respiratory Infections," British Medical Journal, No. 5063, Vol. 1, 1958, pp. 119-128.

Buckland, F. E., and D. A. J. Tyrrell. "Loss of Infectivity on Drying Various Viruses," Nature, Vol. 195, 1962, pp. 1063-1064.

Burke, M. H., H. C. Schenk, and J. A. Thrash. "Tuberculosis Studies in Muscogee County, Georgia. II. X-ray Findings in a Community-Wide Survey and Its Coverage as Determined by a Population Census," Public Health Reports, Vol. 64, 1949, pp. 263-290.

Choudhury, A. K. "Some Epidemiological Considerations of Influenza 1957," Calcutta Medical Journal, Vol. 59, 1961, pp. 231-236. (As quoted in: American Institute of Biological Sciences: An Annotated Bibliography of Influenza with Indexes. Published quarterly. Washington, D.C., 1957-1963.

Chu, C. M., C. H. Andrews, and A. W. Gledhill. "Influenza in 1948-1949," World Health Organization Bulletin, Vol. 3, 1950, pp. 187-214.

Cochrane, A.L., J.G. Cox, and T. F. Jarman. "Pulmonary Tuberculosis in the Rhondda Fach, an Interim Report of a Survey of a Mining Community," British Medical Journal, No. 4789, Vol. 2, 1952, pp. 843-853.

Comstock, G. W. and P. E. Sartwell. "Tuberculosis Studies in Muscogee County, Georgia. IV. Evaluation of a Community-Wide X-ray Survey on the Basis of Six Years of Observation," American Journal of Hygiene, Vol. 61, 1955, pp. 261-285.

Davenport, F. M., and A. V. Hennessy. "A Serologic Recapitulation of Past Experiences with Influenza A; Antibody Response to Monovalent Vaccin," Journal of Experimental Medicine, Vol. 104, 1956, pp. 85-97.

Davenport, F. M., A. V. Hennessy, and T. Francis. "Epidemiologic and Immunologic Significance of Age Distribution of Antibody to Antigenic Variants of Influenza Virus," Journal of Experimental Medicine, Vol. 98, 1953, pp. 641-656.

Davis, W., et al. Development of Typical Urban Areas and Associated Casualty Curves. Albuquerque, N. Mex.: The Dikewood Corporation, 1965.

Davis, W., et al. Prediction of Urban Casualties from the Immediate Effects of a Nuclear Attack. Albuquerque, N. Mex.: The Dikewood Corporation, 1963.

Davis, W., et al. Prediction of Urban Casualties and the Medical Load From a High-Yield Nuclear Burst. Albuquerque, N. Mex.: The Dikewood Corporation, December 1967.

Demographic Yearbook 1963. New York: United Nations, 1964, p. 592. (The Sixth Revision of the International Classification of Diseases is used here.)

"Development and Recommendation of Criteria Needed as a Basis for a National Emergency Medical Care Plan Under Terms of Contract No. CD-SR-58-1." Annex H. Chicago, Ill.: American Medical Association, 1957.

Dohan, F. C. "Air Pollutants and Incidence of Respiratory Disease," Archives of Environmental Health, Vol. 3, 1961, pp. 387-396.

Dowlong, H. W., G. G. Jackson, and T. Inouye. "Transmission of the Experimental Common Cold in Volunteers. II. The Effect of Certain Host Factors Upon Susceptibility," Journal of Laboratory and Clinical Medicine, Vol. 50, 1957, pp. 516-525.

Dubovsky, H. "Mass Miniature X-ray and Tuberculin Survey in Orange Free State and North Cape Colony," South African Medical Journal, Vol. 29, 1955, pp. 992-997.

Eichenwald, H. F., O. Kotsevalov, and L. A. Fasso. "Some Effects of Viral Infection on Aerial Dissemination of Staphylococci and on Susceptibility to Bacterial Colonization," Bacteriological Reviews, Vol. 25, 1961, pp. 274-281.

BIBLIOGRAPHY (Continued)

Francis, T. "A Serological Recapitulation of Human Infection with Different Strains of Influenza Virus," Transactions of the Association of American Physicians, Vol. 66, 1953, pp. 231-239.

Frost, W. H., et al. "Diphtheria in Baltimore, a Comparative Study of Morbidity, Carrier Prevalence and Antitoxic Immuntiy in 1921-1924 and 1933-1936," American Journal of Hygiene, Vol. 24, 1936, pp. 568-585.

Gill, D. G. "Schick Tests and Carrier Surveys in White School Children, Alabama 1937-1938," American Journal of Public Health, Vol. 30 Supplement, 1940, pp. 25-27.

Gordon, J. E. (ed.). Control of Communicable Diseases in Man (9th ed.). American Public Health Association. New York: 1960.

Gordon, John E. Control of Communicable Diseases in Man. New York: The American Public Health Association, 1965.

Grossman, W. "A Schick Test and Diphtheria Carrier Survey of White School Children in Virginia (Richmond)," American Journal of Public Health, Vol. 30 Supplement, 1940, pp. 8-15.

Hallan, J. B., J. L. Colley, W. L. Wells, R. S. Titchen, C. N. Dillard, and A. V. Alhadeff. Review and Evaluation of the National Emergency Health Preparedness Program - Final Summary Report, R-OU-209, Research Triangle Park, N.C.: Research Triangle Institute, 30 November 1966.

Hanegraaf, T. A. Report to the United Nations' Temporary Executive Authority West New Guinea Concerning Tuberculosis Survey in Japan. Unpublished. Hollandia, 1962.

Harper, G. J. "Airborne Micro-Organisms' Survival Test with Four Viruses," Journal of Hygiene, Vol. 59, 1961, pp. 479-486.

Hatch, T. F. "Distribution and Deposition of Inhaled Particles in Respiratory Tract," Bacteriological Reviews, Vol. 25, 1961, pp. 237-240.

Hemmes, J. H., K. C. Winkler, and S. M. Kool. "Virus Survival as a Seasonal Factor in Influenza and Poliomyelitis," Nature, Vol. 188, 1960, pp. 430-431.

Hennessy, A. V., F. M. Davenport, and T. Francis. "Studies of Antibodies to Strains of Influenza Virus in Perons of Different Ages in Sera Collected in a Postepidemic Period," Journal of Immunology, Vol. 75, 1955, pp. 401-409.

Herzog, W. T. Emergency Health Problems Study, Final Report R-OU-106. Research Triangle Park, N.C.: Research Triangle Institute, July 31, 1963.

Hilleman, M. R., et al. "Epidemiology of RI (RI-67) Group Respiratory Virus Infections in Recruit Populations," American Journal of Hygiene, Vol. 62, 1955, pp. 29-42.

Hilleman, M. R., et al. "Epidemiologic Investigations with Respiratory Disease Virus RI-67," American Journal of Public Health, Vol. 45, 1955, pp. 203-210.

BIBLIOGRAPHY (Continued)

Hilleman, M. R. and J. H. Werner, "Recovery of New Agent from Patients with Acute Respiratory Illness," Proceedings of the Society of Experimental Biology and Medicine, Vol. 85, 1954, pp. 183-188.

Isaacs, A., R. J. C. Hart, and V. G. Law. "Influenza Viruses, 1957-1960," World Health Organization Bulletin, Vol. 26, 1962, pp. 253-259.

Jensen, K. E. and T. Francis. "The Antigenic Compostion of Influenza Virus Measured by Antibody Absorption," Journal of Experimental Medicine, Vol. 98, 1953, pp. 619-639.

Jensen, K. E. "The Nature of Serological Relationships Among Influenza Viruses," Advances in Virus Research, Vol. 4, 1957, pp. 279-310.

Johnson, T. and D. R. Johnston. Vectorborne Disease and Control, Final Report R-OU-303. Research Triangle Park, N.C.: Research Triangle Institute, June 1968.

Jordan, W. S., et al. "A Study of Illness in a Group of Cleveland Families, XVII. The Occurrence of Asian Influenza," American Journal of Hygiene, Vol. 68, 1958, pp. 190-212.

Kendall, D. G. "Deterministic and Stochastic Epidemics in Closed Populations," Proceedings: Third Berkeley Symposium on Mathematical Statistics and Probability, Vol. 4. Berkeley and Los Angeles, Calif.: University of California, 1956, pp. 149-165.

Kendall, D. G. "Mathematical Models of the Spread of Infection," Mathematics and Computer Science in Biology and Medicine, pp. 213-225.

Kermack, W. O. and A. G. McKendrick. "A Contribution to the Mathematical Theory of Epidemics," Proceedings of the Royal Society of London, Series A, Vol. 115, 1927, pp. 700-721.

Langmuir, A. D. "Epidemiology of Air-Borne Infection," Bacteriological Reviews, Vol. 25, 1961, pp. 173-181.

Lidwell, O. M. and T. Sommerville. "Observations on the Incidence and Distribution of the Common Cold in a Rural Community During 1948 and 1949," Journal of Hygiene, Vol. 49, 1951, pp. 365-381.

Linder, Forest E. and Robert D. Grove. Vital Statistics Rates in the United States 1900-1940. Washington, D. C.: U. S. Government Printing Office, 1943 pp. 210-215.

Lurie, M. B., et al. "An Evaluation of the Method of Quantitative Air-Borne Infection and Its Use in the Study of the Pathogenesis of Tuberculosis," American Review of Tuberculosis, Vol. 61, 1950, pp. 765-797.

MacLeod, C. M. "Pathogenic Properties of Bacteria and Defense Mechanisms of the Host," Bacterial and Mycotic Infection of Man (3rd edition), (ed. R. J. Dubos). London: Pitman, 1958, pp. 84-113.

McCarroll, J. R., E. J. Cassell, D. W. Wolter, J. D. Mountain, J. R. Diamond, I. M. Mountain. "Air Pollution and Illness in a Normal Urban Population," Archives of Environmental Health, Vol. 14, 1967, pp. 178-184.

McCarroll, J. R., E. J. Cassell, W. I. Ingram, and D. W. Wolter. "Health and the Urban Environment: Health Profiles Versus Environmental Pollutants," American Journal of Public Health, Vol. 56, 1966, pp. 266-275.

Mitchell, H. H. Survey of the Infectious Disease Problem as It Relates to the Postattack Environment, RM-5090-TAB. Santa Monica, Calif.: The RAND Corporation, August 1966.

Mitchell, H. H. Plague in the United States: An Assessment of Its Significance as a Problem Following a Thermonuclear War, RM-4968-TAB. Santa Monica, Calif.: The RAND Corporation, June 1966.

Model 62 Civil Defense Emergency Hospital, Component Listing and Storage Data. Washington, D. C.: Public Health Service, 1964.

Murray, J. F. "Diphtheria Amongst the Bantu," Journal of Hygiene, Vol. 43, 1943, pp. 159-169.

National Office of Vital Statistics. Vital Statistics of the United States, 1964, Vol. 1, Section 1. Washington, D. C.: U. S. Government Printing Office, 1966.

Netherlands' Ministry of Social Affairs. Malnutrition and Starvation. Edited by G. C. E. Burger, J. C. Drummond, and H. R. Sanstead. The Hague: General State Printing Office, 1948.

Pascua, M. "Evolution of Mortality in Europe During the Twentieth Century," World Health Organization Epidemiological and Vital Statistics Report, Vol. 4, 1951, pp. 36-137.

Passive, and Therapeutic Guide for the Civil Defense Emergency Hospital Pharmaceuticals. Washington, D. C.: Public Health Service, 1964.

Rammelkamp, C. H. "Epidemiology of Streptococcal Infections," Harvey Lectures, Vol. 51, 1955-1956, pp. 113-142.

Reed, L. J. and W. H. Frost (1928), as quoted by Abbey in: "An Examination of the Reed-Frost Theory of Epidemics," Human Biology, Vol. 24, 1952, pp. 201-233.

Refsum, E. "Mass Investigations by Photofluroscopy," Acta Tuberculosis Scandinavica, Vol. 27, 1952, pp. 288-302.

Riley, R. L. "Air-Borne Pulmonary Tuberculosis," Bacteriological Reviews, Vol. 25, 1961, pp. 243-248.

Roden, A. T. "Variations in the Clinical Pattern of Experimentally Induced Colds," Journal of Hygiene, Vol. 61, 1963, pp. 231-246.

Sartwell, Phillip E.,(ed.) Maxy-Rosenau: Preventive Medicine and Public Health, 9th edition. New York: Appleton-Century-Crofts, 1965, p. 507.

Schuman, L. M. and J. A. Doull. "Diphtheria Infection and Morbidity in Cleveland, 1937-1939." American Journal of Public Health, Vol. 30 Supplement, 1940, pp. 16-24.

Scrimshaw, N. S., C. E. Taylor, and J. E. Gordon. "Interactions of Nutrition and Infection," American Journal of Medical Sciences, Vol. 237, 1959, pp. 367-403.

Serfling, Robert E. "Historical Review of Epidemic Theory," Human Biology, A Record of Research, Vol. 24, No. 3, pp. 145-166.

Sigurdsson, S. and P. Q, Edwards. "Tuberculosis Morbidity and Mortality in Iceland," World Health Organization Bulletin, Vol. 7, 1952, pp. 153-169.

Simpson, R. E. Hope. "Common Respiratory Diseases--Symposium," Royal Society of Health Journal, Vol. 78, 1958, pp. 593-599.

Simpson, R. E. Hope. "Discussion on the Common Cold," Proceedings of the Royal Society of Medicine, Vol. 51, 1958, pp. 267-271.

Soper, H. E. "Interpretation of Periodicity in Disease Prevalence," Journal of the Royal Statistical Society, Vol. 92, 1929, pp. 34-73.

Statistical Office of the United Nations. Demographic Yearbook, 1963. New York: United Nations, 1964, pp. 592, 609.

Strebbins, E. L. "Diphtheria Immunity and Carrier Surveys in New York State," American Journal of Hygiene, Vol. 30 Supplement, 1940, pp. 36-41.

Stoner, R. D. Radiation and Infection, An Annotated Bibliography. Supplement I. Upton, N. Y.: Medical Research Center Brookhaven National Laboratory, 1967.

Stoner, R. D., M. W. Hess, and V. P. Bond. Radiation and Infection: An Annotated Bibliography. Washington, D. C.: Armed Force Epidemiological Board, Commission on Radiation and Infection, 1965.

Taliaferro, W. H., L. G. Taliaferro, and B. N. Jaroslow. Radiation and Immune Mechanisms. New York: Academic Press, 1964.

Titchen, R. S. Late Post Nuclear Attack Health Problems Study. Research Triangle Park, N. C.: Research Triangle Institute, 1966.

"The Treatment of Mass Civilian Casualties in a National Emergency." Trauma Research Group. Ithaca, N.Y.: Cornell University Medical College, 1964.

Tromp, S. W. "Biometeorological Aspects of Respiratory Diseases." Paper read at Air Pollution Medical Research Conference, Los Angeles, American Medical Association, March 2-4, 1966.

Tuberculosis Survey in the Somalilands (1956), Nigeria (1957), Basutoland, Bechuanaland and Swaziland (1958), Ibadan, Nigeria (1958), Ghana (1958), Uganda (1959), Sierra Leone (1959). Copenhagen: World Health Organization Tuberculosis Research Office (year of publication as indicated above).

U. S. Bureau of the Census. Mortality Statistics. Washington, D. C.: U. S. Government Printing Office, published annually, 1901-1936.

U. S. Bureau of the Census. Vital Statistics of the U. S. Washington, D.C.: U. S. Government Printing Office, published annually, 1937-1949.

U. S. Dept. Of Health, Education, and Welfare. International Classification of Diseases, Adapted. Washington, D. C.: Public Health Service, 1962.

Van Loghem, J. J. "An Epidemiological Contribution to the Knowledge of the Respiratory Diseases," Journal of Hygiene, Vol. 28, 1928, pp. 33-54.

Vital Statistics of the United States 1950, Vol. I, Table 8-45. Washington, D. C.: U. S. Government Printing Office, p. 218.

Vital Statistics of the United States 1964, Vol. I. Table 1-6. Washington, D. C.: U. S. Government Printing Office, p. 1-6.

Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Endemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina. Doctoral dissertation, 1965.

Walker, William. "The Aberdeen Typhoid Outbreak of 1964," Scotland Medical Journal, 1956, pp. 466-79.

Webster, L. T. "Experimental Epidemiology," Medicine, Vol. 25, 1946, pp. 77-109.

Wells, W. L. and W. J. Cromartie. Shelter Medical Support System Study. Research Triangle Park, N. C.: Research Triangle Institute, 1963.

Wijsmuller, G. Tuberculosis Survey Reports to the Government of Netherlands, New Guinea. Unpublished. Hollandia, 1956-1961.

Wijsmuller, G. Naturally Acquired Tuberculin Sensitivity in New Guinea. Thesis. Amsterdam: 't Koggeschip, 1963.

Wiles, F. J. and C. J. Rabie. "Tuberculin and X-ray Surveys in the Transkey," South African Medical Journal, Vol. 29, 1955, pp. 866-868.

Williams, T. "The Basic Birth-Death Model for Microbial Infections," The Journal of the Royal Statistical Society. Series B, Vol. 27, No. 2, 1965.

Woodall, J., K. C. K. Rowson, and J. C. McDonald. "Age and Asian Influenza," British Medical Journal, No. 5108, Vol. 2, 1958, pp. 1316-1318.

Appendix A

Glossary of Terms and Symbols for the Total Health Care System Model

The purpose of this glossary is to introduce a precise definition for those terms and symbols used to describe the Total Health Care System Model. These definitions are provided separately for the two submodels of this Total Model; i.e., The Immediate Effects Submodel and the Disease and Chronic Condition Submodel.

Appendix A

Glossary of Terms and Symbols for the Total Health Care System Model

I. GLOSSARY OF TERMS FOR THE IMMEDIATE EFFECTS SUBMODEL

Allied Medical Personnel. A team consisting of a combination of two or more personnel trained as nurses, pharmacists, veterinarians or dentists.

Casualty. Designation restricted here to injured persons who reach the emergency treatment center alive.

Casualty Spectrum (Injury Spectrum). The list of injury types and the number of casualties reported for each.

Downgrading. Assigning a batch of casualties to be treated by personnel at the next lower level of treatment. For example, under some conditions casualties with injuries for which the preferred level is the physician level may be treated by allied medical personnel, or injuries that should be treated by surgical teams might be treated by physicians.

Emergency Treatment Center. A station established in each subdivision (grid) of an attack area not having a central hospital. All casualties in each of these subdivisions are collected at the Emergency Treatment Center for triage and either treated in the Center or transferred to the central hospital. The hospital also serves as an Emergency Treatment Center for the subdivision in which it is located.

First Phase. See Initial Treatment Phase.

Follow-on Treatment. The medical attention given during the time period after the initial treatment time period to casualties who have or have not received initial treatment in the Initial Treatment Phase. For the purposes of allocating resources, physicians are assumed to work one-half time or 12 hours per day.

Golden Period. The period immediately following the attack in which delay in treatment does not cause the probability of death to rise.

Grid. One of the subdivisions of the community, each of which contains one Emergency Treatment Center.

Hospital. The central hospital of a given district. It may represent a composite of several hospitals treated as a unit. The hospital serves as the Emergency Treatment center for the subdivision in which it is located. The hospital is the only place which provides treatment by skilled surgical teams.

Immediate Effects. Damage and injury inflicted by the blast, thermal radiation, and gamma radiation of the weapon at the moment of detonation and immediately thereafter.

Immediate Effects Recovery Phase. The second part of the Immediate Effects Submodel, this phase involves (a) follow-on treatment for those surviving casualties who received initial treatment, and (b) treatment of additional casualties brought in after the initial treatment phase.

Initial Treatment Phase. The first phase of the Immediate Effects Submodel, this phase is concerened with the triage and intital treatment of victims of the immediate effects of the weapon. (Physicians work 24 hours a day.)

Injury Caseload. Number of casualties with a particular injury.

Medical Resources. Medical resources consist of treatment personnel and medical supplies. Treatment personnel are further classified in the model as physicians (all physicians, including osteopaths, regardless of specialty) or as allied medical personnel; i.e., professionals, excepting physicians, associated with the medical field. For example, dentists, veterinarians, nurses, and x-ray technicians would be considered as allied medical personnel. Medical supplies are those expendable items used in treating injuries; i.e., drugs, dressings, splints, fluids, etc.

Medical Supply Package. (Also referred to as "Medical Treatment Package"). A kit designed to provide all medical supplies necessary to treat one casualty suffering from a particular injury. There may be as many kinds of packages as there are injuries, or they may be designed so that one type of package is appropriate to more than one type of injury. Sixteen medical treatment packages required for classes of injuries have been derived based on the MEND[1] recommendations.

Physician. A team consisting of a physician and one or more assistants.

Preferred Treatment Level. Refers to the level of treatment associated with each injury. Casualties will be given treatment at this level except when personnel are unavailable or when a better prognosis will result from earlier treatment by personnel at the next lower level.

Priority for Treatment. The priority assigned to the several injury categories determines the order in which the batches of casualties will be attended to. The highest priority is "1" and the lowest is "9". Since priority is independent of level of treatment, injuries within each are taken in order sequentially according to the code numbers of the injuries.

Surgical Team. A team consisting of at least (a) one surgeon, (b) one anesthetist, and (c) two nurses (or equivalent).

Survivors. Consists of surviving uninjured and casualties processed by either an Emergency Treatment Center or hospital.

Treatment Facilities. Treatment facilities are of two types; i.e., hospital and emergency treatment facilities. The hospital facilities include operating rooms, specialized personnel, and medical supplies. General and specialized hospitals, and the 200-bed Packaged Disaster Hospitals correspond to this facility definition.[2] Emergency treatment facilities may be located at a stocked fallout shelter, a physician's office, a medical arts building, or a drug store.

---

[1] "The Treatment of Mass Civilian Casualties in a National Emergency," Trauma Research Group. Ithaca, N.Y.: Cornell University Medical College, 1964.

[2] "Austere Medical Care for Disaster." Washington, D.C.: U.S. Public Health Service, 1964.

Treatment Level. The level of treatment corresponds to the category of personnel administering it. The four treatment levels considered in the Initial Treatment Phase are given as follows:

a) No Treatment (level 1): No medical personnel are assigned at this level although simple custodial and supervisory care should be available to keep patients in this treatment level (those whose prognosis is poor) as comfortable as possible and to allow them to attend basic body needs.

b) Surgical Team (level 2): A team consisting of at least (1) one surgeon, (1) one anesthetist, and (3) two nurses (or equivalent).

c) Physician (level 3): A physician and an assistant or assistants.

d) Allied Medical Personnel (level 4): A team of two or more nurses, pharmacists, veterinarians, or personnel trained in self-help.

Treatment Personnel. Treatment personnel are defined under "Medical Resources" above.

Triage. The sorting of incoming casualties into groups according to treatment level. Each injury in this model has assigned to it a preferred level of treatment, but in the process of triage, the actual level of treatment may be changed according to rules governing different circumstances.

II. GLOSSARY OF TERMS AND SYMBOLS FOR THE DISEASE AND CHRONIC CONDITIONS SUBMODEL

Symbol or Unit of Study	Definition
A	Crude attack rate, expressed as number of individuals per unit of time.
α	The proportion of infected individuals that will become infective.
αD	This product is the average number of days of infectivity of an infected host ("the index of lack of nonspecified host resistance" or, less accurately, "the nonspecific susceptibility).
B	Secondary attack ratio, expressed as the proportion of the community members that is secondarily attacked.
Contact	An adequate contact is the exchange of breath and/or respiratory tract secretions between two individuals that is long and intensive enough to transmit the disease if one of the two individuals involved is susceptible and the other infective.
D	Average duration (expressed in time units) of infectivity in a host who has become infective. (Also used in subscripts, see t).
d	This symbol, placed before a quantity, indicates the amount of change in this quantity during a very short time.
e	Base of the Naperian (natural) logarithm with the constant value 2.71828...
I	The number of infective individuals in the community.
I_t	The number of infective individuals in the community at time t.
$i = \frac{I}{N}$	The prevalence of infectives in the community, expressed as a proportion.
λ	The rate (number of persons per unit of time) at which a community member establishes contact with the other susceptible, infective, or immune members. This rate is named contact rate.
λ'	The rate (number of individuals per time unit) at which a community member establishes contacts with the other susceptible, infective, or immune members of his household.

Symbol or Unit of Study	Definition
λ''	The rate (number of individuals per time unit) at which a community member establishes contacts with other susceptible, infective, or immune members outside of his household.
M	The number of births per time unit in the community.
$m = \frac{M}{N}$	Birth rate of the community, expressed as a proportion per unit of time.
N	The total number of individuals (members) in the community.
n	The number of individuals in a household.
o	The subscript o indicates the "equilibrial" or "homeostatic" value towards which the mean value of a time series of the relevant characteristic moves, if this mean is taken over a sufficiently long time. In stochastic models where there is a stationary process, the equivalent is the "expected value."
Pr(XY/Z)	The probability of the intersection of events X and Y, given event Z.
p	The probability of at least one contact between any two specified individuals in the community during one unit of time.
p'	The probability of at least one contact between any two specified individuals in the same household within the community during one unit of time.
p''	The probability of at least one contact between any two specified individuals belonging to different households within the community during one unit of time.
$q = 1 - p$	The probability of no contact between any two specified individuals in the community during one time unit.
$q' = 1 - p'$	The probability of no contact between any two specified individuals, belonging to the same household, during one unit of time.
$q'' = 1 - p''$	The probability of no contact between any two specified individuals belonging to different households within the community, during one unit of time.
S	The number of susceptible individuals in the community.
S_t	The number of susceptible individuals in the community at time t.

Symbol or Unit of Study	Definition
$s = \frac{S}{N}$	The prevalence of susceptibles in the community expressed as a proportion.
t	The subscript t refers to the point in time to which the relevant subscripted characteristic is applicable (see for instance S_t below).
Unit of Time	Unless otherwise specified, the time unit is the time interval between infection and beginning of infectivity ("the incubation period").
Z	The number of immune individuals in the community.
Z_t	The number of immune individuals in the community at time t.

Appendix B

Injury Prognoses and Treatment Times in Disaster Medicine

This appendix describes the data elements representing the prognoses and treatment parameters for each injury class. These data form part of the input for the Immediate Effects Submodel.

Appendix B

Injury Prognoses and Treatment Times in Disaster Medicine

I. INTRODUCTION

Emergency health preparedness includes such programs as the stockpiling of essential medical items, the development and deployment of federally subsidized Packaged Disaster Hospitals, and the training of professionals and nonprofessionals in emergency medical care. Because of the number and complexity of variables associated with emergency health preparedness, it was necessary to use a simulation modeling approach to study the immediate and late effects of a disaster. In the time sense used here, "immediate" implies roughly from time zero to two weeks and is primarily concerned with injured casualties and their care. Late effects are associated with the time period from two weeks to about one year and include problems associated with acute and chronic diseases.

II. PURPOSE OF THIS APPENDIX

A necessary input to the Immediate Effects Simulation Model is a description of the injuries suffered by surviving casualties, along with the probable fate of each injury category when afforded a specified treatment after a specified delay. This Appendix (B) outlines the data which were derived for each injury in order to simulate emergency medical care.

Associated with each injury is a prognosis as a function of time delay in treatment, which takes the general form shown in Figure B-1. The time from injury to a time, T_1, is a period during which delay in treatment will not affect the prognosis. After T_1, further delays in treatment are accompanied by a linear increase in probability of death. At some time, T_2, the prognosis has deteriorated to P_{nt}, the prognosis associated with no treatment.

Since little quantative data are available, the prognosis data used was obtained from a local medical consultant, Dr. Warner Wells. Since the simulation can accept data from other sources as well, it is expected that the effect of different notions about disaster prognoses may be tested in future studies. The relative magnitude of differences between upper and lower bounds on fatalities for a given caseload are directly related to the differences between prognoses under ideal or no treatment conditions.

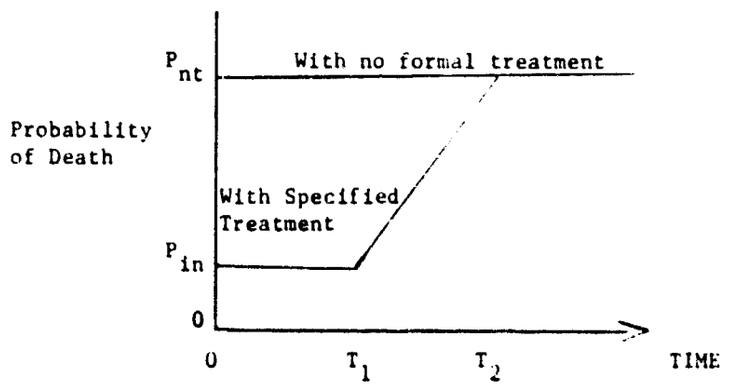


Fig. B-1. Form of the Injury Prognosis Curve

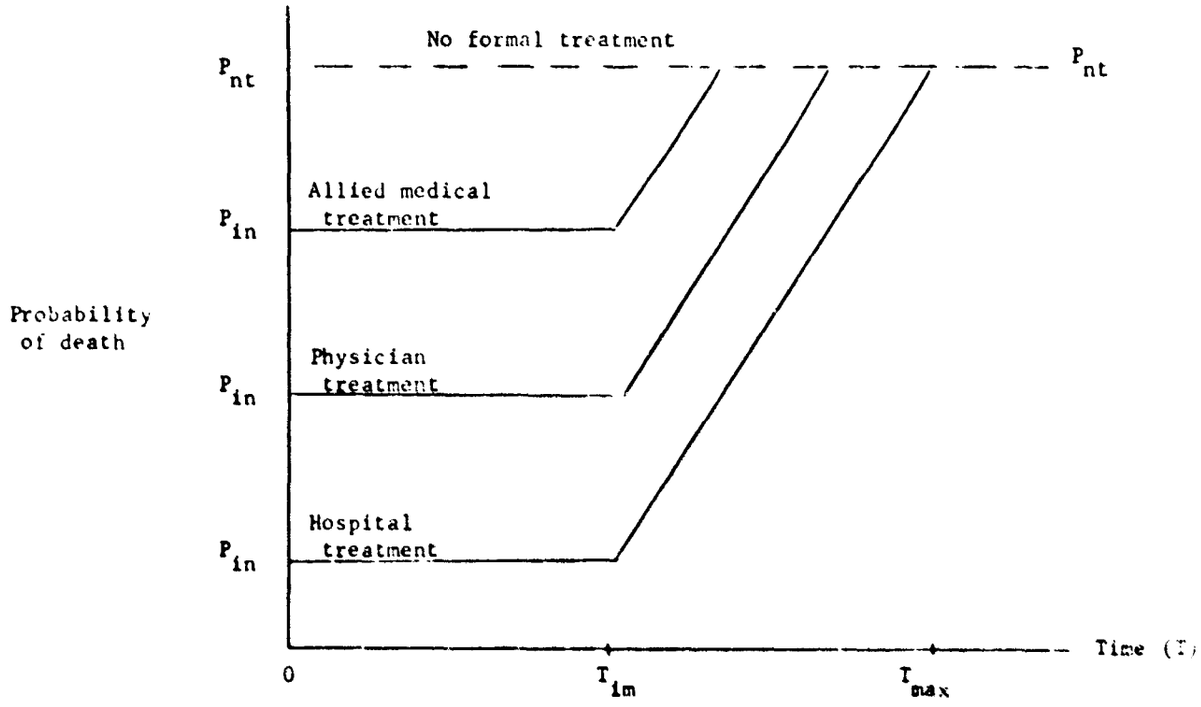


Fig. B-2. Injury Prognosis for Different Treatment Levels

III. DESCRIPTION OF DATA ELEMENTS

A. Nature of Injury

Little data are available that relates specific injuries to thermonuclear weapon parameters; consequently, the injury spectrum used in this study is largely a reflection of plausible reasoning and judgment. Of chief importance in the initial selection of the injury spectrum was an earlier study of Wells and Cromartie[1/] in which an illness and injury spectrum of a model community was examined in terms of survival with varying degrees of medical austerity.

In this baseline study, four categories of injury severity (ranging from severe to minor) were used to classify injuries juxtaposed with differing levels of medical care. The current study includes injuries from only the two most serious categories, i.e.,

(a) Category I - injuries which could not be adequately treated except in a hospital; and

(b) Category II - injuries which could be treated by physicians and assistants with drugs, dressings and other equipment outside a hospital.

Also of considerable importance in defining the injury spectrum were early historical findings, the atomic bombings of Japan, the Texas City, Texas disaster, and the experience gained by Dr. Wells while in residence at Hiroshima, Japan during the Atomic Bomb Casualty Commission Studies. The injuries used as input to the model are categorized in the manner described above, i.e., the most severe injuries will be found in Category I and those of a lesser severity in Category II. In addition, the injuries may be further divided into 7 major classifications:

1. Mechanical
2. Burn
3. Pure Radiation
4. Mechanical-burn injury combination
5. Mechanical injury and specified radiation dose
6. Burn injury and specified radiation dose
7. Mechanical-burn injury combination with specified radiation dose.

1/ Wells, W. L. and W. J. Cromartie. Shelter Medical Support System Study. Research Triangle Park, N. C.: Research Triangle Institute, 1963.

Specific injury titles follow the nomenclature used in the International Classification of Diseases, Adapted[2/] (ICDA). The mechanical-burn injury combinations are not as specific and do not follow the ICDA nomenclature. It was decided to consider combination injuries only in broad terms of severity; i.e., severe mechanical-severe burn, severe mechanical-moderate burn, moderate mechanical-severe burn, and moderate mechanical-moderate burn. The severe burn or mechanical injury component of a combination injury denotes a severity such as that found in Category I injuries (described above). A moderate burn or mechanical injury component of a combination injury is indicative of the severity of injuries found in Category II injuries.

B. Prognoses Data

The probability of death, or prognosis, for each injury has been estimated by Dr. Wells using available clinical data and judgment. The ICDA nomenclature (mentioned above) has figured heavily in these estimates. As used in ICDA, each injury title in the sample may actually be indicative of several types of specific lesions which might occur to the anatomical region, organ, or skeletal structure mentioned. For instance, "skull fracture" as used in the sample includes fractures to the vault of the skull and the base of the skull. Fractures of long bones, such as the femur or tibia as used in the sample, are indicative of both closed and open fractures. Internal injuries to the organs within the chest and abdomen are also nonspecific. That is, an injury to the gastrointestinal tract is representative of damage to the stomach, duodenum, intestine, colon, etc., which may have resulted from severe contusions, hematoma, or tearing. In each case, the prognosis for the injury stated in the table is indicative of the average prognosis for all injuries within that ICDA grouping. The relative severity of the injuries is reflected by their placement within one of the two categories discussed above. Obviously, these prognoses data are not precise. They are, however, reasonable and plausible first approximations to mass disaster injury prognoses.

Historical findings were also important in the development of the prognosis data. In order to provide baseline estimates of injury prognoses under austere conditions, it was necessary to examine in some detail historical data associated with physical injury, woundings, and impairments.

2/ International Classification of Diseases, Adapted, U. S. Department of Health, Education and Welfare. Washington, D. C.: Public Health Service, 1962

Data are also presented on each injury as further complicated by LD_{50} and LD_{25} doses of whole body radiation. Data on LD_{50} doses were provided by Dr. Wells. For a specified injury, LD_{25} data are linear interpolations between the LD_{50} data and the data considering no radiation dose. Injuries complicated by radiation doses above LD_{50} were not considered because of the very high probability of death.

The various prognoses have been determined for three levels of treatment and as a function of time delay to treatment. These take the form shown in Figure B-2. The various treatment options and the preferred level of treatment are discussed in detail in Sections III, C and D of this Appendix.

Each injury prognosis at a specified treatment level includes the following factors:

1. P_{in} (Probability of Death at $T \leq T_{im}$) - an estimate of the probability that a casualty chosen at random from all of the same type will die if given early specified treatment. The probability of death (P_{nt}) has also been estimated, assuming the injury receives no formal treatment.
2. T_{im} (Effective Treatment Time) - a period of time during which there is essentially no change in probability of mortality because of delay in treatment. T_{im} is necessarily the same for all levels of treatment.
3. T_{max} (Ineffectual Treatment Time) - the time after which the probability of death with a specified treatment is equal to the probability of death with no formal treatment. For a given injury, T_{max} has been arbitrarily set to be numerically equal to twice T_{im} for all levels of treatment. The relative importance of this approximate rule has been determined. Test runs with varying values of T_{max} indicate that it is a critical parameter in assessing injury survivors (see Chapter 2). It is recognized that no ratio would be equally applicable to all injuries; it is to be emphasized that the T_{max} data currently employed represents a compromise which has allowed model development work to continue.

C. Treatment Level

Associated with each injury are several treatment levels, of which one is "preferred" (defined subsequently). These treatment levels, which were described by Dr. Wells in an earlier study, include:

1. No Formal Treatment Level

   Casualties with injuries classified as "1" are either beyond help or so slightly injured that treatment is not required for survival.

2. Surgical Team Level

Treatment at this level is of the highest quality. It consists of facilities and staff corresponding to those of a fully-equipped hospital, including operating rooms, surgical teams, and supplies. This level also includes the 200-bed Packaged Disaster Hospitals.

3. Physician Level

Treatment at this level is administered by, or under the direction of, a physician at a hospital or an emergency treatment center. Physicians, other personnel (allied medical[3/] and medical self-help[4/]), and specified medical supplies and equipment are located at such treatment centers. The location of centers was determined by the locations of medical supplies, i.e., at fallout shelters, physicians' offices, nursing homes, drug stores, etc.

4. Allied Medical Personnel

This level is reserved for treatment which can be administered by allied medical personnel or those trained in "medical self-help" utilizing austere supplies such as those stocked in public fallout shelters. This treatment can take place in a hospital as well as in an emergency medical treatment center. This level represents the lowest quality of formal treatment available.

D. Preferred Level of Treatment

As indicated above, one of the treatment level options is preferred over the others. This preferred level is the lowest level of care which can be administered for a specified injury without significantly jeopardizing survival. Because radiation injury cannot be readily diagnosed in its very early stages, the preferred levels are chosen for the nonirradiated injuries and then assigned to the LD_{50} and LD_{25} cases also.

3/ The Public Health Service has defined categories of allied health personnel to include such professions as dentistry, nursing, veterinary medicine, etc. For detailed information consult: "Austere Medical Care for Disaster", p. iii. Washington, D. C.: U. S. Public Health Service, 1964.

4/ Medical self-help courses are designed to teach the public the rudiments of emergency medical care in the absence of more skilled personnel. "Family Guide Emergency Health Care," U. S. Department of Health, Education, and Welfare. Washington, D. C.: U. S. Public Health Service, 1965.

The preferred level of treatment for a specified injury was empirically derived on the basis of differences between the probabilities of death at various levels of treatment. It appeared obvious that if relatively insignificant differences existed between the probabilities of death at the surgical level of treatment and a lower level, then the lower level of treatment would be preferred. Presumably this would prevent the flooding of the more sophisticated care levels with cases not necessarily worthy of their attention. Thus, certain empirical rules were established which convey this attitude. These rules, in their order of application, are as follows:

1. If the difference in the initial probabilities of death (P_{in} in Figure B-2) between the (a) surgical team and no formal treatment levels is $\leq .20$ and (b) allied medical teams and no formal treatment levels is $\leq .10$, the preferred treatment level is that of no formal treatment. If both of these conditions are not met, the preferred level of treatment is one of the highest levels.
2. If the difference in the initial probabilities of death between the (a) surgical team and no formal treatment levels is $\leq .20$ and (b) allied medical teams and no formal treatment levels is $\geq .10$, the allied medical team level is preferred. The allied medical team level is also preferred if the differences in initial probabilities of death between the surgical team level and the allied medical personnel level is $\leq .15$. If this difference is $\geq .15$, the preferred level of treatment is some higher level.
3. If the difference in the initial probabilities of death between the surgical team level and the physician level is $\leq .10$, the physician level is the preferred level of treatment. If the difference is $> .10$, the preferred level is the surgical team level.

Other methods might be employed to determine the preferred level of treatment. One straightforward way would be stipulation by some medical authority. Ultimately, however, alternative determinations of preferred levels of treatment should be the object of sensitivity analysis.

In the immediate effects simulation model, the level of treatment for a given injury may be different from the preferred level based on various decision rules which come into play when critical resources are depleted. These decision rules are described in Chapter 2, III. C 2.

E. Priority of Treatment

Associated with each injury is a specified priority for treatment based upon the principles of triage. Triage is a classification scheme to effectively utilize limited medical resources in treatment of mass casualties. There are a number of methods whereby priorities for treatment among various injuries can be determined. Any method for assigning priorities for treatment must take into account the utilization of scarce resources (personnel and supplies) and the anticipated return from their utilization.

The method used in this study is as follows: Once a preferred level of treatment has been determined for a given injury, the increase in probability of death if the injury were to receive the next lower level of care (P_{in} preferred - P_{in} lower level) is determined. Injuries which incur a large increase in probability of death as a result of downgraded care should be given high priority at the preferred level. If the preferred level is "No Formal Treatment" level, the lowest priority is assigned.

If personnel treatment time requirements (T_T) are low for an injury category, this category should have a high priority.

A third factor considered is the period of time (T_{im}) during which there is no change in probability of mortality due to treatment delay. Injured who have short T_{im} values should be among the first treated. Thus, a high-priority patient can be thought of as one having minimal T_{im}'s and T_T's and a maximum ΔP_ℓ as the result of downgrading to the next lower level of treatment. These factors have been arranged to provide a numerical basis for priority determination as follows:

1. Obtain a Treatment Priority Index:

$$\text{Treatment Priority} = \frac{\Delta P_\ell}{T_{im} + T_T}$$

2. Rank all injuries in decending order on the basis of the priority index; i.e., highest treatment priority index first, next highest second, etc. All injuries with a preferred level of "no treatment" are assigned a priority index of 0.
3. The ranked list of injuries with nonzero treatment priority indexes are divided uniformly into eight groups. The first eighth of this ranked list assigned a treatment priority of 1, the second eighth a treatment priority of 2, etc. All injuries with a priority index of 0 are assigned to a treatment priority 9.

It should be noted that supplies required for treatment should also be considered in determining priorities, but a more sophisticated level of priority determination was not possible within the time frame of model development. However, other methods of determining priorities can also be used in terms of types and amounts of supplies required for treatment and treatment time.

The treatment priority for an injured person is based only on the injury since the influence of radiation exposure is not accurately known. The treatment priority so calculated is then used for the LD_{50} and the LD_{25} cases as well.

F. Treatment Time

Associated with the treatment of each injury at a specified level is the time required for treatment. This time is not the total man-hours required. For instance, at the surgical team level a treatment time of three hours represents three hours of physician time and three hours each for the other members of a four-man surgical team. The same is true for the physician who is assisted by one allied medical person (or someone trained in medical self-help) and for the two-man teams of allied medical personnel or persons trained in medical self-help. Treatment times and prognoses associated with each treatment level were estimated by Dr. Wells.

IV. INJURY DESCRIPTION AND PROGNOSIS DATA

A. Introductory Comments

The following table contains a listing of the representative injuries used in the simulation model. An associated prognosis, treatment priority, preferred treatment level are given for each injury. Treatment times are given for the three levels of formal treatment.

As indicated previously, the Category I injuries are those whose changes of survival are relatively poor unless afforded sophisticated care. For instance, such injuries as a pelvic fracture may be open and may involve the ilium, ischium, pubic bone, or acetabulum. The fracture may have been caused by a missile or a crushing blow and may be further complicated by infection. The femur fracture as used here is generally considered to be compound and possibly complicated by infection. The brain contusion is indicative of an open intracranial wound with the possible complication of infection. Injuries to such internal organs as the spleen, kidney, G. I. tract, and pelvic organs are indicative of that which might result from a foreign body with the associated problem of infection. Severe contusion, hematoma, or tearing are also considered present in the injuries to the abdomen and thorax.

The probable outcome of the Category II injuries is relatively simple to estimate except perhaps for the fracture of the tibia/fibula and ankle. These fractures are considered open and complicated by infection.

Since the various injury categories often reflect several aspects of one injury, their ICDA Code numbers (International Classification of Diseases, Adapted) are not mutually exclusive. For instance, a skull fracture (800) may be accompanied by fracture of face bones (802) and by concussion (852).

In many instances, downgrading of the treatment level was judged not to increase the probability of death (P_{in}) even if the downgraded time of treatment (T_T) became shorter. Such a downgrading, however, may cause permanent disability that is not reflected in the table.

B. Table of Injuries and Prognoses Data

(See pages B-11 - B-20.

Table B-I

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLDEN PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

	NATURE OF INJURY	ICDA Code No.[1]	PTL[2]	TP[3]	T_{im}[4] (hrs)	TREATMENT LEVELS[5] Surg. P_{in}	Surg. T_T (hrs)	Phys. P_{in}	Phys. T_T (hrs)	AM P_{in}	AM T_T (hrs)	NT P_{nt}
CATEGORY 1 INJURIES	Skull fracture	800-803; 850-856	3	6	12	0.25	2.00	0.35	1.00	0.50	0.50	0.90
	Skull fracture - LD_{50} dose	Ditto	3	6	12	0.75	2.00	0.85	1.00	1.00	0.50	1.00
	Fracture of vertebra (cord injury)	805-806	2	6	12	0.25	2.25	0.50	1.00	0.50	0.50	0.95
	Fracture of vertebra (cord injury) LD_{50} dose	Ditto	2	6	12	0.75	2.25	1.00	1.00	1.00	0.50	1.00
	Pelvis fracture	808; 867-867.0	2	6	6	0.10	2.00	0.35	1.00	0.50	0.50	0.75
	Pelvis fracture - LD_{50} dose	Ditto	2	6	6	0.60	2.00	0.85	1.00	1.00	0.50	1.00
	Fracture of femur	820-821.5; 890-890.2	3	3	6	0.10	1.60	0.20	1.00	0.40	0.75	0.60
	Fracture of femur - LD_{50} dose	Ditto	4	1	6	0.60	1.60	0.70	1.00	0.70	0.75	1.00
	Brain contusion	853-855	3	3	12	0.10	2.00	0.20	0.50	0.30	0.50	0.75
	Brain contusion - LD_{50} dose	Ditto	2	8	12	0.60	2.00	0.70	0.50	0.80	0.50	0.90
	Heart or lung injury	807; 860-862.1	2	5	6	0.25	2.70	0.75	0.75	0.75	0.50	0.95
	Heart or lung injury LD_{50} dose	Ditto	3	3	6	0.75	2.70	1.00	0.75	1.00	0.50	1.00
	G. I. tract	863-863.1	2	2	6	0.50	2.25	0.75	0.75	0.75	0.50	0.95
	G. I. tract - LD_{50} dose	Ditto	2	7	96	0.55	2.25	1.00	0.75	1.00	0.50	1.00
	Kidney injury	866-866.1	2	2	6	0.50	2.25	0.75	0.75	0.75	0.50	0.90
	Kidney injury - LD_{50} dose	Ditto	2	7	96	0.55	2.25	1.00	0.75	1.00	0.50	1.00
	Spleen injury	865-865.1	2	2	6	0.50	2.25	0.75	0.75	0.85	0.50	0.90
	Spleen injury - LD_{50} dose	Ditto	2	7	96	0.55	2.25	1.00	0.75	1.00	0.50	1.00
	Injury to pelvic organs	867-867.1; 879	2	4	6	0.10	2.25	0.60	0.75	0.65	0.50	0.90
	Injury to pelvic organs - LD_{50} dose	Ditto	2	7	6	0.60	2.25	1.00	0.75	1.00	0.50	1.00

Table B-I (Con't)

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLDEN PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

	NATURE OF INJURY	ICDA Code No.[1]	PTL[2]	TP[3]	T_{im}[4] (hrs)	TREATMENT LEVELS[5] Surg. P_{in}	Surg. T_T (hrs)	Phys. P_{in}	Phys. T_T (hrs)	AM P_{in}	AM T_T (hrs)	NT P_{nt}
CATEGORY I INJURIES	Chest and abdomen	860-869.1; 879	2	4	6	0.25	2.50	0.75	0.75	0.75	0.50	0.95
	Chest and abdomen - LD_{50} dose	Ditto	1	9	96	0.90	2.50	1.00	0.75	1.00	0.50	1.00
	Foreign body entering through orifice, pharynx and larynx	933	2	3	6	0.25	1.00	0.50	1.00	0.75	0.50	0.95
	Foreign body entering through orifice, pharynx and larynx - LD_{50} dose	Ditto	2	3	6	0.75	1.00	1.00	1.00	1.00	0.50	1.00
	Foreign body through bronchus and lungs	934	2	7	6	0.25	2.70	0.50	1.00	0.75	0.50	0.95
	Foreign body through bronchus and lungs - LD_{50} dose	Ditto	2	7	6	0.75	2.70	1.00	1.00	1.00	0.50	1.00
	Burns over 20-30% of body	940-949	2	7	6	0.10	2.00	0.25	0.75	0.30	0.50	0.50
	Burns over 20-30% of body - LD_{50} dose	Ditto	4	1	6	0.75	2.00	0.75	0.75	0.80	0.50	1.00
	Burns over 30-40% of body	940-949	2	7	6	0.30	2.50	0.50	0.80	0.60	0.60	0.75
	Burns over 30-40% of body - LD_{50} dose	Ditto	1	9	6	0.90	2.50	1.00	0.80	1.00	0.60	1.00
	Burns over 40-50% of body	940-949	2	6	6	0.55	2.50	0.90	0.80	0.95	0.60	0.98
	Burns over 40-50% of body - LD_{50} dose	Ditto	1	9	6	1.00	2.50	1.00	0.80	1.00	0.60	1.00
	Burns over more than 50% of body.	940-949	1	9	6	0.80	2.50	0.97	0.25	0.98	0.25	0.99
	Burns over more than 50% of body - LD_{50} dose	Ditto	1	9	6	1.00	2.50	1.00	0.25	1.00	0.25	1.00

Table B-I (Con't)

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLD. ' PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

	NATURE OF INJURY	ICDA Code No.[1]	PTL[2]	TP[3]	T_{im}[4] (hrs)	TREATMENT LEVELS[5] Surg. P_{in}	Surg. T_T (hrs)	Phys. P_{in}	Phys. T_T (hrs)	AM P_{in}	AM T_T (hrs)	NT P_{nt}
CATEGORY I INJURIES	Severe blast and severe thermal	800-898; 940-949	1	9	6	0.75	4.00	0.90	0.25	0.90	0.25	0.95
	Severe blast and severe thermal LD_{50} dose	Ditto	1	9	6	1.00	4.00	1.00	0.25	1.00	0.25	1.00
	Severe blast and moderate thermal	800-898; 940-949	2	8	6	0.25	4.00	0.40	0.25	0.55	0.25	0.70
	Severe blast and moderate thermal - LD_{50} dose	Ditto	1	9	6	0.80	4.00	0.90	0.25	1.00	0.25	1.00
	Moderate blast and severe thermal	800-898; 940-949	2	8	6	0.65	4.00	0.80	0.25	0.90	0.25	0.99
	Moderate blast and severe thermal - LD_{50} dose	Ditto	1	9	6	1.00	4.00	1.00	0.25	1.00	0.25	1.00
	Moderate blast and moderate thermal	800-898; 940-949	1	9	6	0.22	3.00	0.30	0.50	0.30	0.50	0.35
	Moderate blast and moderate thermal - LD_{50} dose	Ditto	1	9	6	0.80	3.00	0.85	0.50	0.90	0.50	0.95
	Radiation dose - LD_{50}	990	1	9	96	0.50	0.50	0.50	0.50	0.50	0.50	0.50
	Radiation dose - LD_{75}	990	1	9	96	0.75	0.50	0.75	0.50	0.75	0.50	0.75
CATEGORY II INJURIES	Head concussion	852	1	9	12	0.00	2.00	0.01	0.50	0.05	0.50	0.10
	Head concussion - LD_{50} dose	Ditto	1	9	96	0.50	2.00	0.50	0.50	0.55	0.50	0.60
	Eye and orbit laceration	870-871	1	9	6	0.05	1.30	0.10	1.00	0.20	0.75	0.25
	Eye and orbit laceration - LD_{50} dose	Ditto	1	9	6	0.55	1.30	0.60	1.00	0.70	0.75	0.75
	Foreign body through orifice, eye and adnexa	870-871	4	2	24	0.05	1.00	0.10	1.00	0.10	0.50	0.25
	Foreign body through orifice, eye and adnexa - LD_{50} dose	Ditto	4	2	24	0.55	1.00	0.60	1.00	0.60	0.50	0.75

Table B-I (Con't)

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLDEN PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

	NATURE OF INJURY	ICDA Code No. 1/	PTL 2/	TP 3/	T_{im} 4/ (hrs)	TREATMENT LEVELS 5/						
						Surg.		Phys.		AM		NT
						P_{in}	T_T (hrs)	P_{in}	T_T (hrs)	P_{in}	T_T (hrs)	P_{nt}
CATEGORY II INJURIES	Laceration and open wound of shoulder and upper arm	880-880.2	1	9	6	0.00	1.00	0.00	0.50	0.00	0.50	0.05
	Laceration and open wound of shoulder and upper arm - LD_{50} dose	Ditto	1	9	96	0.50	1.00	0.50	0.50	0.50	0.50	0.60
	Laceration and open wound of elbow, forearm, wrist	881-881.2	1	9	6	0.00	1.00	0.00	0.50	0.00	0.50	0.05
	Laceration and open wound of elbow forearm, wrist - LD_{50} dose	Ditto	1	9	96	0.50	1.00	0.50	0.50	0.50	0.50	0.60
	Laceration of hand	883-883.2	1	9	6	0.00	1.00	0.00	0.50	0.00	0.15	0.05
	Laceration of hand - LD_{50} dose	Ditto	1	9	96	0.50	1.00	0.50	0.50	0.50	0.15	0.60
	Laceration of hip and thigh	890-890.2	1	9	6	0.00	1.00	0.00	0.50	0.00	0.30	0.05
	Laceration of hip and thigh - LD_{50} dose	Ditto	1	9	96	0.50	1.00	0.50	0.50	0.50	0.30	0.60
	Laceration of knee, leg, foot, toes	891-891.2	1	9	6	0.00	1.00	0.00	0.50	0.00	0.20	0.05
	Laceration of knee, leg, foot, toes - LD_{50} dose	Ditto	1	9	96	0.50	1.00	0.50	0.50	0.50	0.20	0.60
	Rib fracture	807-807.0	1	9	6	0.00	1.50	0.05	1.00	0.08	0.75	0.10
	Rib fracture - LD_{50} dose	Ditto	3	7	96	0.50	0.50	0.60	1.00	0.70	0.75	0.75
	Clavicle fracture	810-810.0	1	9	24	0.01	0.50	0.01	0.50	0.01	0.50	0.02
	Clavicle fracture - LD_{50} dose	Ditto	1	9	96	0.55	0.50	0.55	0.50	0.55	0.50	0.60
	Humerus fracture	812-812.0	1	9	24	0.01	1.00	0.03	0.50	0.03	0.50	0.10
	Humerus fracture - LD_{50} dose	Ditto	4	1		0.55	1.00	0.55	0.50	0.55	0.50	0.75
	Fracture of radius and/or ulna	813-813.0	1	9	24	0.01	1.00	0.03	0.50	0.03	0.50	0.10
	Fracture of radius and/or ulna - LD_{50} dose	Ditto	4	1	96	0.55	1.00	0.55	0.50	0.50	0.50	0.75

Table B-1 (Con't)

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLDEN PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

	NATURE OF INJURY	ICDA Code No. [1]	PTL [2]	TP [3]	T_{im} [4] (hrs)	TREATMENT LEVELS [5]						
						Surg.		Phys.		AM		NT
						P_{in}	T_T (hrs)	P_{in}	T_T (hrs)	P_{in}	T_T (hrs)	P_{nt}
CATEGORY II INJURIES	Fracture of hand bones (multiple)	814-814.0; 817-817.1	1	9	24	0.01	1.00	0.01	0.50	0.01	0.50	0.02
	Fracture of hand bones (multiple) - LD_{50} dose	Ditto	1	9	96	0.55	1.00	0.55	0.50	0.55	0.50	0.55
	Fracture of tibia and/or fibula	823-823.0	4	6	6	0.03	1.30	0.10	1.00	0.15	0.75	0.25
	Fracture of tibia and/or fibula - LD_{50} dose	Ditto	4	6	96	0.50	1.30	0.60	1.00	0.65	0.75	0.75
	Ankle fracture	824-824.0	4	5	6	0.03	1.30	0.10	1.00	0.15	0.50	0.25
	Ankle fracture - LD_{50} dose	Ditto	4	3	96	0.50	1.30	0.60	1.00	0.65	0.50	0.75
	Tarsal metatarsal fracture	825-825.0	1	9	6	0.03	1.25	0.05	0.75	0.10	0.50	0.10
	Tarsal metatarsal fracture - LD_{50} dose	Ditto	1	9	96	0.50	1.25	0.55	0.75	0.65	0.50	0.70
	Dislocation of jaw	830-830.0	4	1	6	0.00	0.50	0.00	0.50	0.00	0.50	0.25
	Dislocation of jaw - LD_{50} dose	Ditto	4	1	96	0.50	0.50	0.50	0.50	0.50	0.50	0.75
	Dislocation of shoulder	831-831.0	1	9	6	0.00	0.50	0.00	0.50	0.00	0.50	0.00
	Dislocation of shoulder - LD_{50} dose	Ditto	1	9	96	0.50	0.50	0.50	0.50	0.50	0.50	0.50
	Dislocation of elbow	832-832.0	1	9	6	0.00	1.25	0.00	0.50	0.00	0.50	0.00
	Dislocation of elbow - LD_{50} dose	Ditto	1	9	96	0.50	1.25	0.50	0.50	0.55	0.50	0.60
	Dislocation of wrist	833-833.0	1	9	6	0.00	1.25	0.00	0.50	0.00	0.50	0.00
	Dislocation of wrist - LD_{50} dose	Ditto	1	9	96	0.50	1.25	0.50	0.50	0.50	0.50	0.60

Table B-I (Con't)

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLDEN PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

NATURE OF INJURY	ICDA Code No.[1/]	PTL [2/]	TP [3/]	T_{im} [4/] (hrs)	TREATMENT LEVELS [5/] Surg. P_{in}	Surg. T_T (hrs)	Phys. P_{in}	Phys. T_T (hrs)	AM P_{in}	AM T_T (hrs)	NT P_{nt}
CATEGORY II INJURIES											
Dislocation of finger	834-834.0	1	9	6	0.00	0.50	0.00	0.25	0.00	0.25	0.00
Dislocation of finger LD_{50} dose	Ditto	1	9	96	0.50	0.50	0.50	0.25	0.50	0.25	0.50
Dislocation of knee	836-836.0	1	9	6	0.05	0.10	0.10	0.50	0.10	0.50	0.12
Dislocation of knee - LD_{50} dose	Ditto	1	9	96	0.60	1.00	0.60	0.50	0.60	0.50	0.75
Open scalp wound	850-850.0	1	9	6	0.01	0.50	0.02	0.50	0.03	0.25	0.06
Open scalp wound - LD_{50} dose	Ditto	4	1	96	0.50	0.50	0.55	0.50	0.60	0.25	0.75
Laceration and open wound of face and neck	873-873.0; 874-874.0	1	9	6	0.00	0.75	0.00	0.50	0.00	0.50	0.05
Laceration and wound of face and neck - LD_{50} dose	Ditto	1	9	96	0.50	0.75	0.50	0.50	0.50	0.50	0.60
Laceration and open wound of chest	875-875.0	1	9	6	0.00	1.00	0.00	1.00	0.00	1.00	0.05
Laceration and open wound of chest - LD_{50} dose	Ditto	1	9	96	0.50	1.00	0.50	1.00	0.50	1.00	0.60
Laceration and open wound of back	876-876.0	1	9	6	0.00	0.50	0.00	0.50	0.00	0.50	0.05
Laceration and open wound of back - LD_{50} dose	Ditto	1	9	96	0.50	0.50	0.50	0.50	0.50	0.50	0.60
Burns over less than 1% of body	940-949	1	9	6	0.00	0.50	0.00	0.25	0.00	0.25	0.01
Burns over less than 1% of body - LD_{50} dose	Ditto	1	9	96	0.60	0.50	0.60	0.25	0.60	0.25	0.70
Burns over 19-20% of body	940-949	1	9	6	0.02	1.00	0.10	0.25	0.10	0.25	0.15
Burns over 19-20% of body - LD_{50} dose	Ditto	1	9	96	0.60	1.00	0.65	0.25	0.70	0.25	0.75

Table B-1 (Con't)

PROBABILITY OF DEATH AND TREATMENT TIME, BY INJURY (WITH SPECIFIED PREFERRED TREATMENT LEVEL, TREATMENT PRIORITY AND "GOLDEN PERIOD"), BY TREATMENT LEVEL, AND BY TREATMENT CATEGORY

CATEGORY II INJURY	NATURE OF INJURY	ICDA Code No. 1/	PTL 2/	TP 3/	T_{im} 4/ (hrs)	TREATMENT LEVELS 5/						
						Surg.		Phys.		AM		NT
						P_{in}	T_T (hrs)	P_{in}	T_T (hrs)	P_{in}	T_T (hrs)	P_{nt}
	Radiation dose - LD_{25}	990	1	9	96	0.25	0.50	0.25	0.50	0.25	0.50	0.25

Source: Wells, W. L. Unpublished data (RTI-OU-209).

1/ International Classification of Diseases, op. cit.

2/ PTL = Preferred Treatment Level

3/ TP = Treatment Priority

4/ T_{im} = Time during which delay in treatment does not alter initial probability of Mortality.

5/ Treatment Level Key:

Surg. = Surgical Team Treatment Level
Phys. = Physician Treatment Level
AM = Allied Medical Treatment Level
NT = No Formal Treatment Level
P_{in} = Initial Probability of Death with Specified Treatment
T_T = Treatment Time in Hours
P_{nt} = Probability of Death with No Treatment

Appendix C

Total Emergency Health Care System Model Flow Charts

This appendix contains flow charts of the basic logic underlying the overall flow of the Total Emergency Health Care System Model (Figure C-1), the basic flow of the Immediate Effects Submodel (Figure C-2), the expanded flow of the Immediate Effects Submodel (Figure C-3), and the basic flow of the Disease and Chronic Conditions Submodel (Figure C-4).

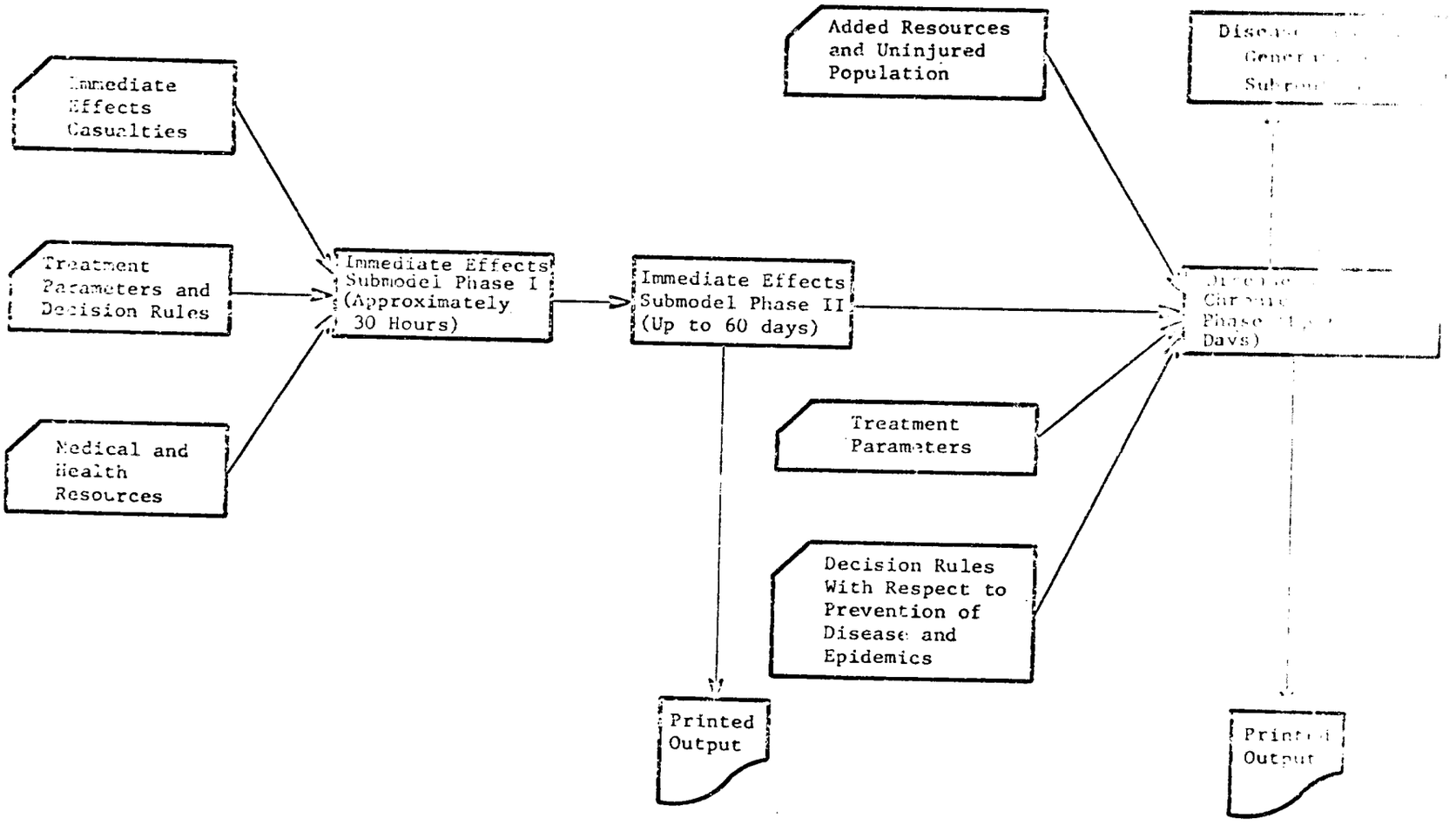


Fig. C-1. Overall Flow of Total Emergency Health Care System Model

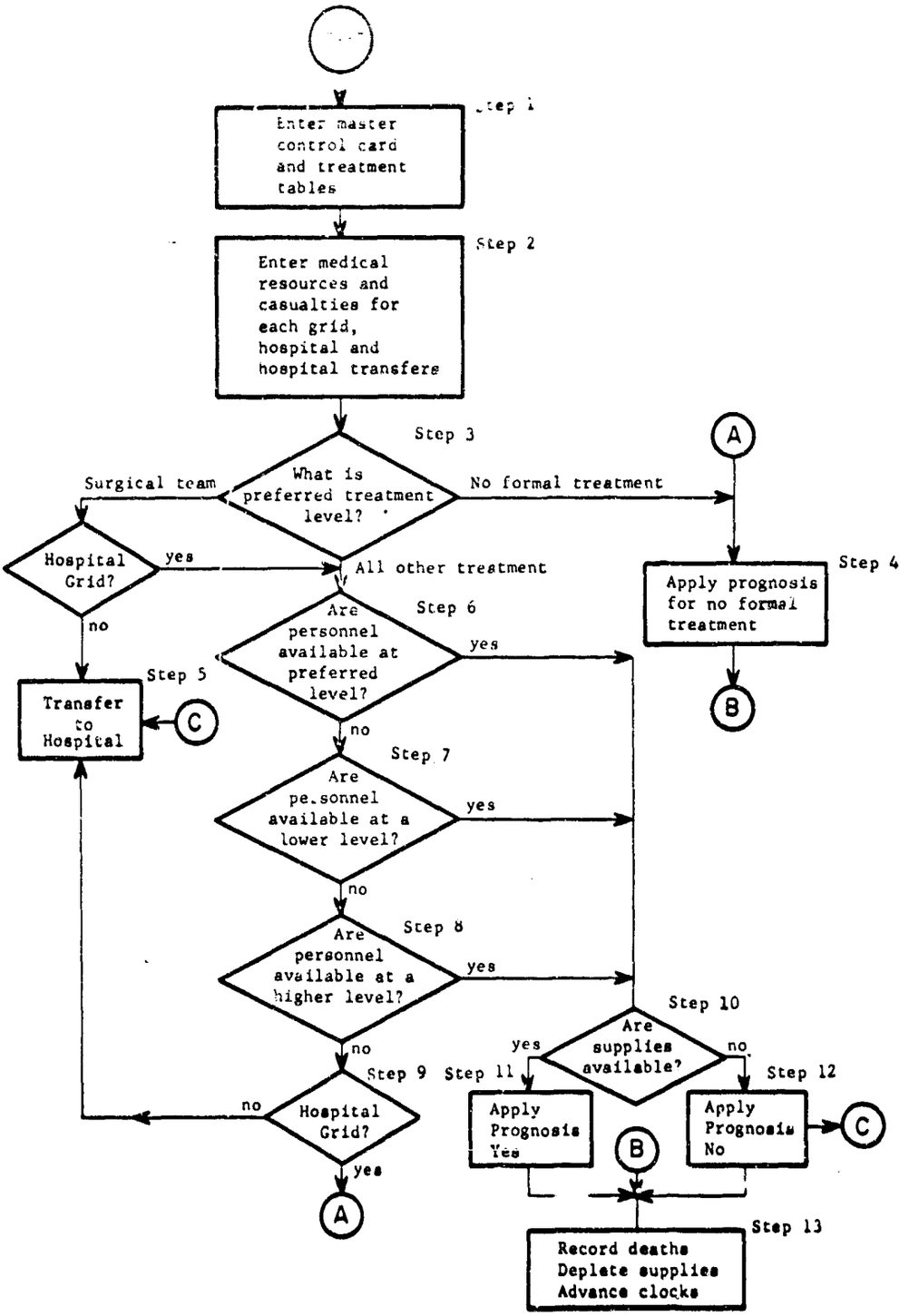


Fig. C-2. Overall Flow of the Immediate Effects Submodel

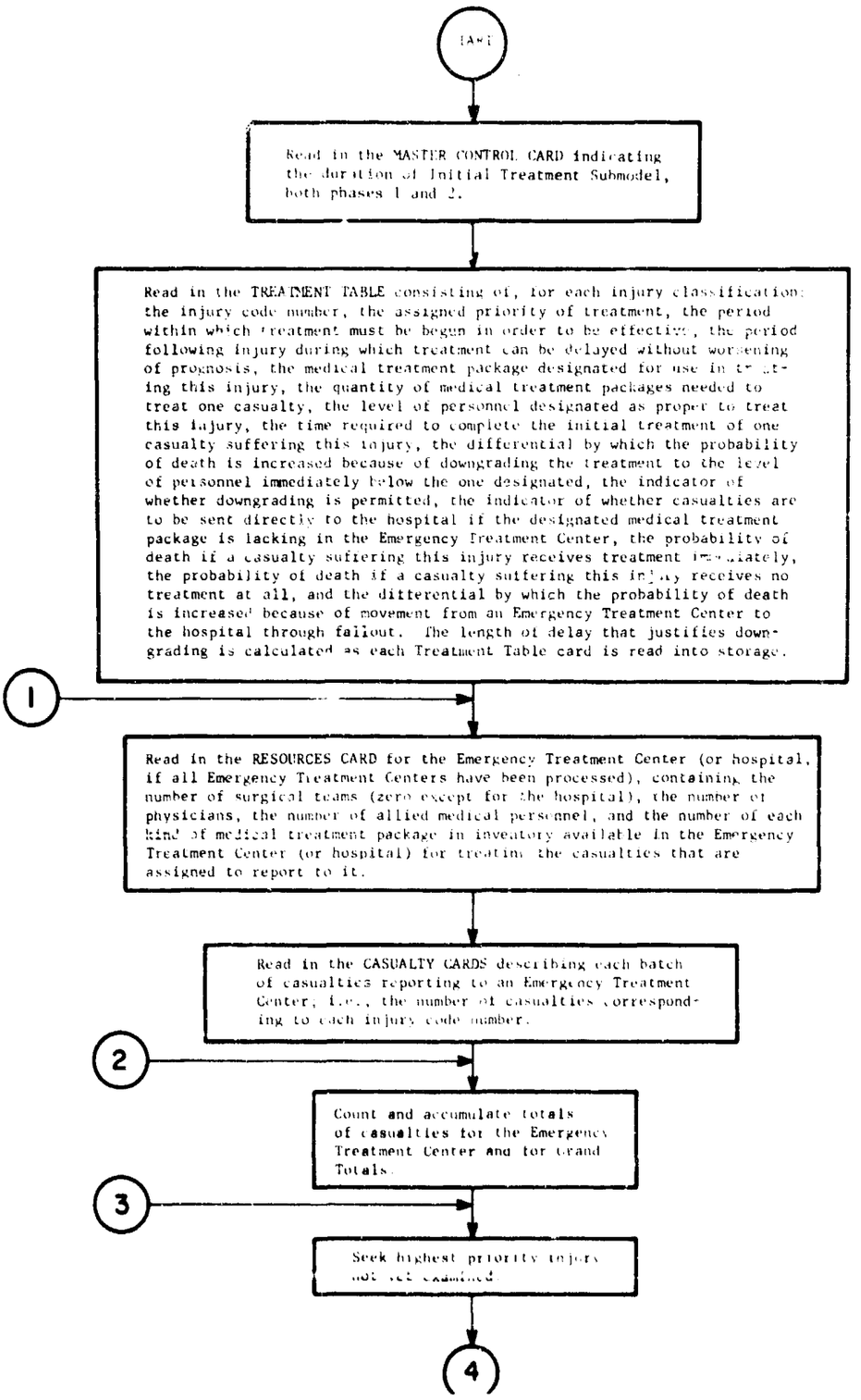


Fig. C-3. Expanded Flow of the Immediate Effects Submodel Logic

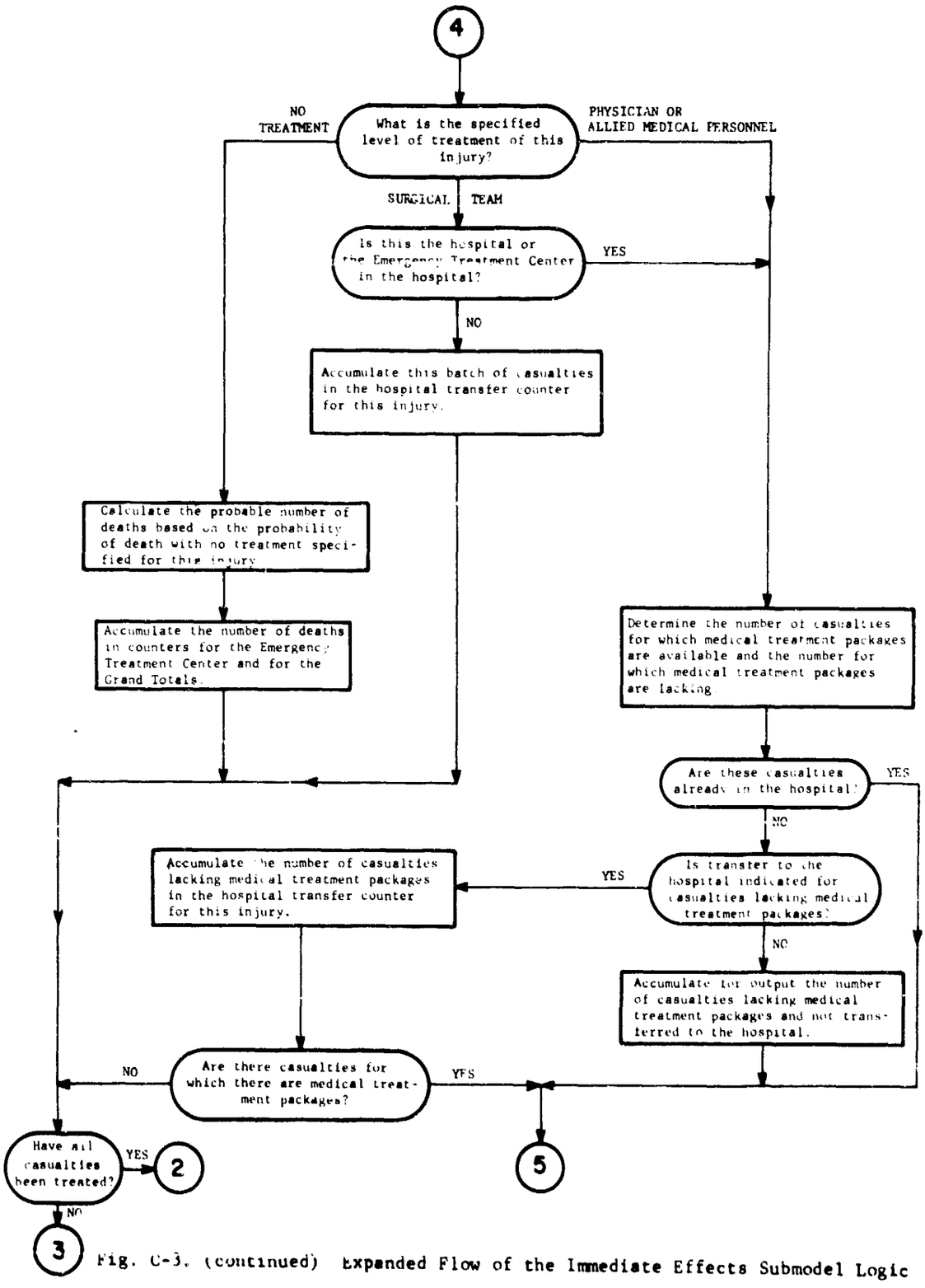


Fig. C-3. (continued) Expanded Flow of the Immediate Effects Submodel Logic

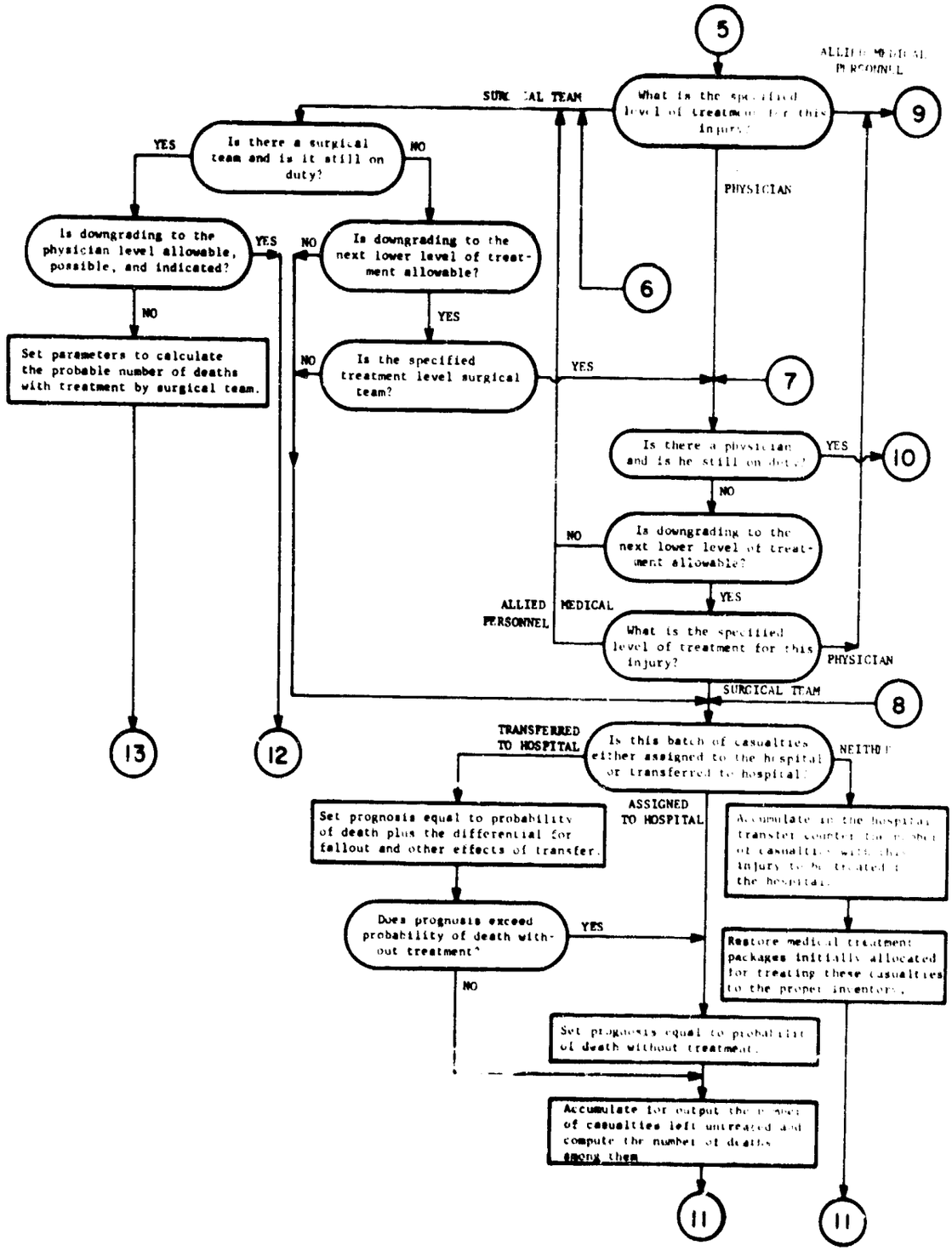


Fig. C-3. (continued) Expanded Flow of the Immediate Effects Submodel Logic

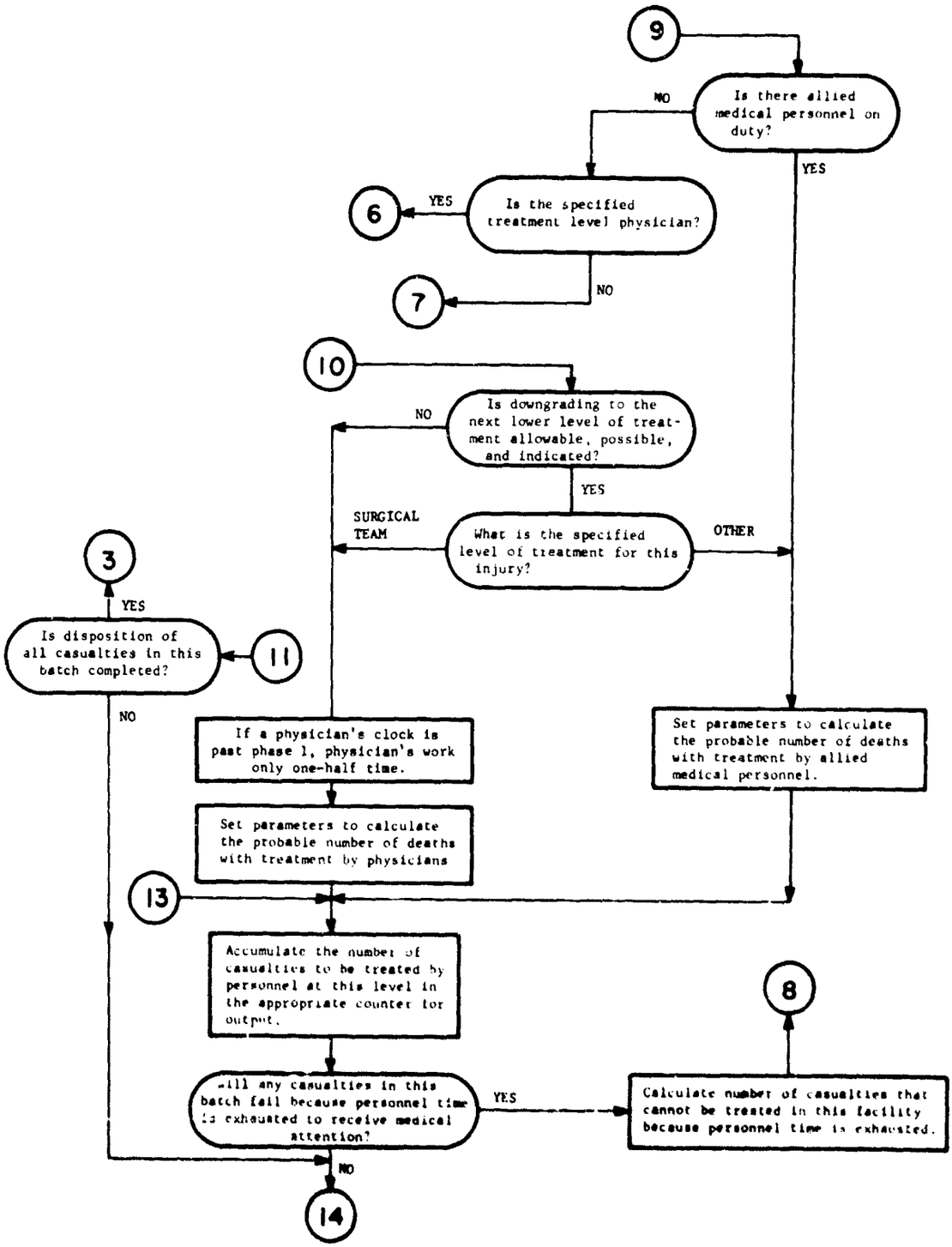


Fig. C-3. (continued) Expanded Flow of the Immediate Effects Submodel Logic

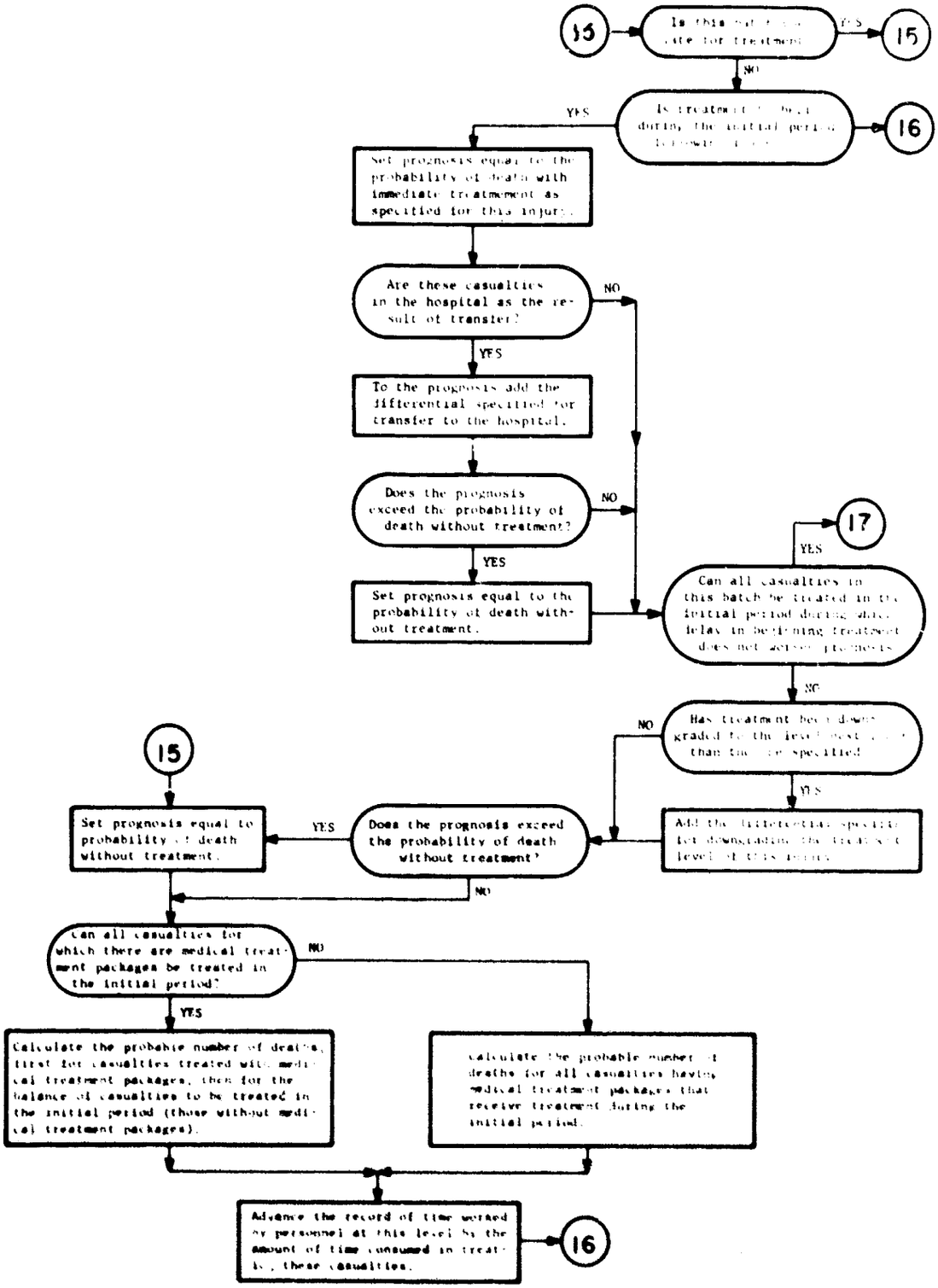


Fig. C-3. (continued) Expanded Flow of the Immediate Effects Submodel Logic

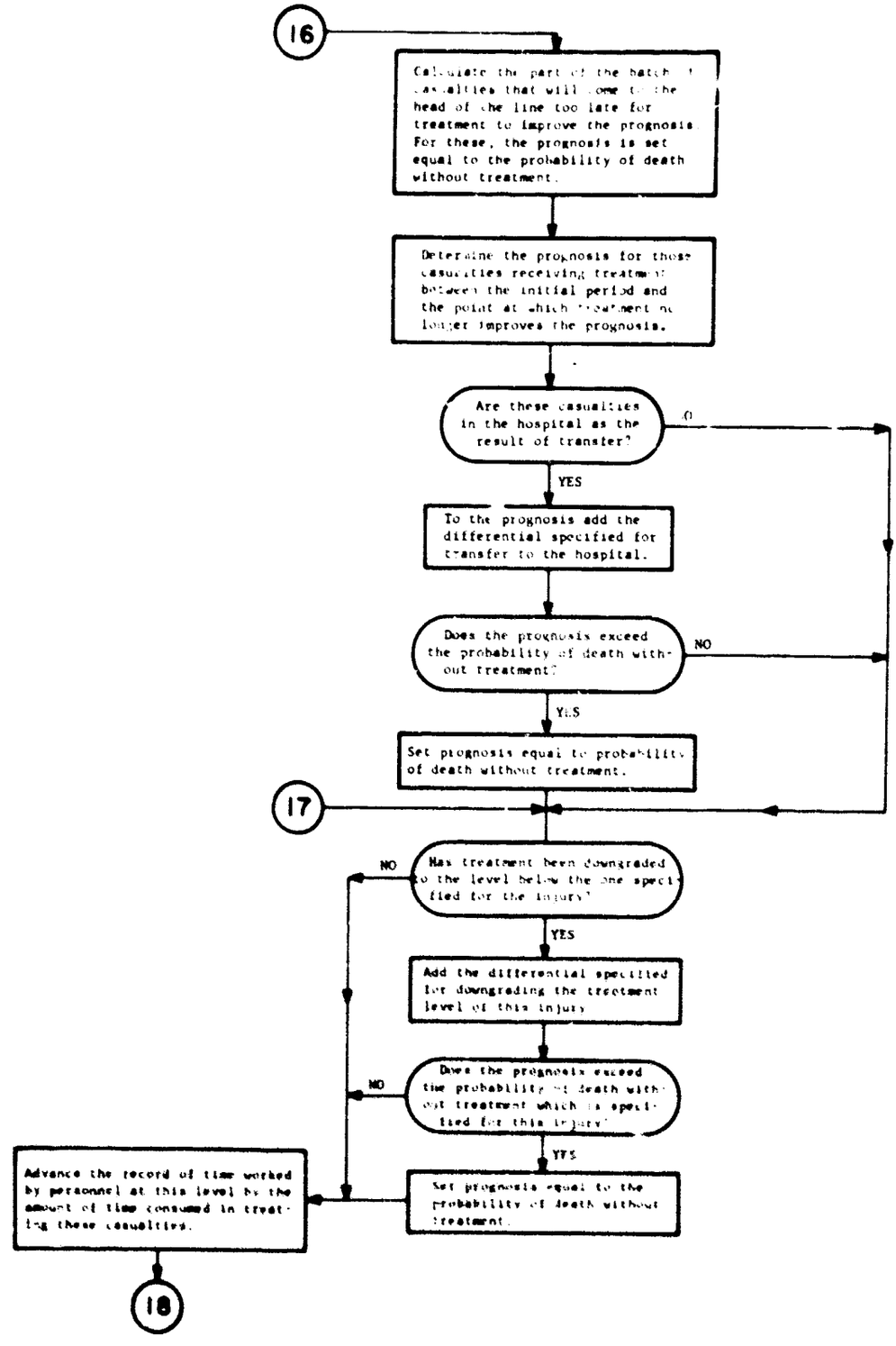


Fig. C-3. (continued) Expanded Flow of the Immediate Effects Submodel Logic

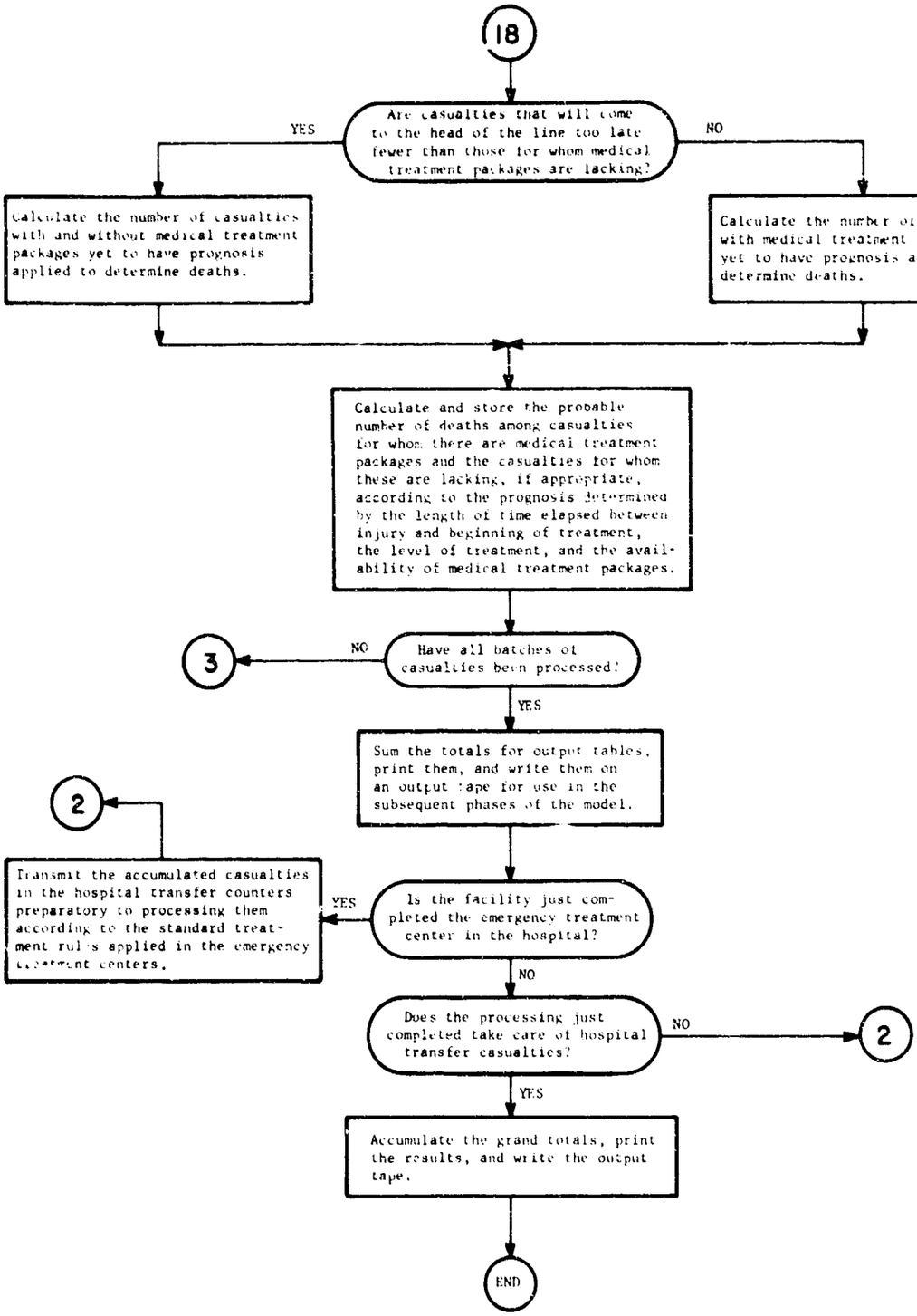


Fig. C-3. (continued) Expanded Flow of the Immediate Effects Submodel Logic

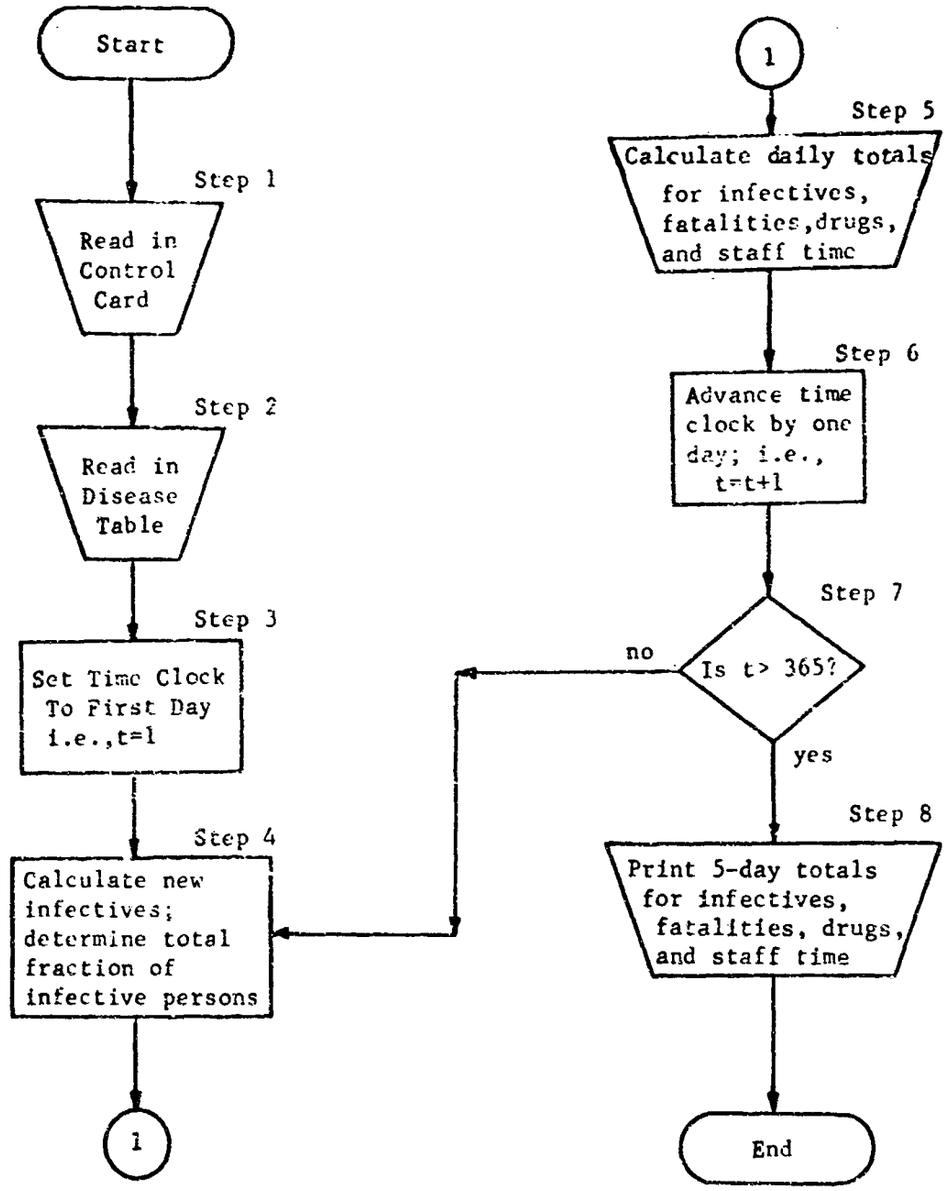


Fig. C-4. Overall Flow of the Disease and Chronic Conditions Submodel

Appendix D

Total Emergency Health Care System Model Data Inputs and Outputs

This appendix presents in complete detail the description and definitions of all data elements used in the design of the Total Emergency Health Care System Model.

Appendix D

Total Emergency Health Care System Model
Data Inputs and Outputs

I. INTRODUCTION

The purpose of this appendix is to present in complete detail the descriptions and definitions of all data elements used in the design of the Total Emergency Health Care System Model. An attempt is made to relate the data elements to their precise uses in the model logic. The appendix is divided into four subsections:

II. Card Inputs to the Immediate Effects Submodel

III. Card Inputs to the Disease and Chronic Conditions Submodel

IV. Outputs from the Immediate Effects Submodel

V. Outputs from the Disease and Chronic Conditions Submodel

The descriptions of the card inputs for each of the phases include the names, definitions, external formats, and maximum values for all data set elements. The descriptions of the outputs include all of the format and indexing options anticipated, and, when appropriate, sample print-outs illustrating the options.

The Master Control Card contains information common to both submodels of the Total Health Care System Model.

Parameter	Card Columns	Definition
NCC	1-6	Card Code = 100000
INPTIM	7-9	Duration in hours for Phase 1 of the Immediate Effects Submodel
INITIM	12-14	Duration in days for Phase 2 of the Immediate Effects Submodel
ITØTIM	17-19	Duration in weeks of Disease and Chronic Conditions Phase

II. CARD INPUTS TO THE IMMEDIATE EFFECTS SUBMODEL

This subsection contains descriptions of all input data sets which can be input to the Immediate Effects Submodel. All the data sets are required for the operation of the model, and the values must appear on the punched cards as specified. Included in the description of each input data set are:

1) The data set name.
2) The submodel using the data set.
3) A general description--one or two sentences describing the data set and the information that it contains.
4) The name of each data set element--six or less alphanumeric characters used to designate the particular data set element.
5) The index, where applicable, indicated in parentheses immediately following the data set element name.
6) The definition of each data set element.
7) The positions on the punched card of each data set element.
8) The field specification (in Fortran notation) of each data set element on the punched card.

The remainder of this subsection is devoted to presenting the individual input data sets.

Input Data Set Number II-1

Data Set Name: Run Identification

Submodel Using Data Set: Immediate Effect Submodel

General Description: A card containing data that describe the particular computer run which are printed at the beginning of the set of tables for each treatment center.

Data Set Elements:

Name	Definition	Card Column	Format
NCC	Card code. For the Run Identification card it is "110000".	1-6	I6
	Heading to identify the computer run (e. g., city name and resources level).	21-80	60H

Input Data Set Number II-2

Data Set Name: Treatment Table

Submodel Using Data Set: Immediate Effects Submodel

General Description: A set of cards containing treatment rules, times, probabilities, and other necessary data for each different injury functioning as a table of variable decisions applicable through a series of runs.

Data Set Elements:

Name	Definition	Card Column	Format
NCC	Card code. For the Injury Treatment Table it is "1".	1	I1
NPRI	Priority of Treatment ("1" through "9").	2	I1
INJRY	Three-digit number designating the injury ("1" through "999").	3-5	I3
XTME	Period following which treatment no longer decreases the probability of death (in hours).	7-8	F2.0
TIM	Initial period during which delay in beginning treatment does not increase the probability of death (in hours).	9-12	F4.0
NDRG	Number designating the medical treatment package for the injury.	13-14	I2
KTY	Quantity of medical treatment packages required to treat one casualty with this injury.	15-16	I2
NTRMT	Level of personnel designated to treat the injury.	17	I1
TIME	Time required for initial treatment of one casualty (in hours).	18-21	F4.2
DPLVL	Increase in probability of death due to downgrading.	22-25	F3.2
LDOWNG	Downgrade flag: if "0", downgrading is allowed; if "3", downgrading is not allowed (when personnel of appropriate level are unavailable or when the prognosis would be better with earlier treatment by personnel at the next lower level).	30	I1

Input Data Set Number II-2 (Continued)

Name	Definition	Card Column	Format
NFLGI	Hospital flag: if "0", treat without medical treatment package and if "1", transfer to the hospital (when supply of necessary package is exhausted).	31	I1
PDIT	Probability of death if treatment is received immediately after the injury.	35-38	F4.2
PDNT	Probability of death if no treatment at all is received.	39-42	F4.2
DRAD	Increase in probability of death due to transportation to hospital, travel through fallout, etc.	51-54	F4.2
LASTCD	Last card indicator: the digit "9" indicates the last card of the Injury Treatment Table.	80	I1

Input Data Set Number II-3

Data Set Name: Emergency Treatment Center Resources

Submodel Using Data Set: Immediate Effects Submodel

General Description: A card read for each treatment center in sequence giving the assigned resources of personnel and medical treatment packages.

Data Set Elements.

Name	Definition	Card Columns	Format
NCC	Card code. For the Resources Card it is "2".	1	I1
NGRDA	Identification number of the emergency treatment center (1 through 998) or hospital (always 999).	2-4	I3
NCD	Secondary card code. For the Resources Card it is "00".	5-6	I2
NSRGT	Number of surgical teams (found only in the hospital), maximum 999.	7-9	I3
NPHYS	Number of physicians, maximum 999.	10-12	I3
NALLD	Number of allied medical personnel, maximum 9,999.	13-16	I4
NPACK(I)	Number of each type of medical treatment package, maximum of each 9,999. There may be up to 16 different medical treatment packages.	21-24 etc.	I4

Input Data Set Number II-4

Data Set Name: Emergency Treatment Center Casualty Report

Submodel Using Data Set: Immediate Effects Submodel

General Description: Cards read for each treatment center immediately after the Emergency Treatment Center Resources card listing by injury number the number of casualties suffering from each injury. Up to 99 Casualty Report cards may be entered for each treatment center. Each Casualty Report card may contain figures for a maximum of ten injury types, allowing a total of 990 for the treatment center. (Program dimensions at present allow for only 150 injury types.) Each card must be filled before proceeding to the next one, since the program ceases to store data on encountering the first blank injury number. Injury numbers may be entered in any order.

Data Set Elements:

Name	Definition	Card Columns	Format
NCC	Card code. For the Casualty Report it is "2".	1	I1
NGRDC	Identification number of the emergency treatment center (1 through 9) or hospital (always 999).	2-4	I3
NCD	Secondary card code. For the Casualty Report card it is "01" through "99".	5-6	I2
INJI(I)	Injury number corresponding to one in the Injury Treatment Table.	10-13 etc.	I4
NXI(I)	Number of casualties with injury specified by INJI(I).	7-9 etc.	I3

III. CARD INPUTS TO THE DISEASE AND CHRONIC CONDITIONS SUBMODEL

This subsection contains descriptions of both input data sets which can be input to the Immediate Effects Submodel; i.e, the control card and the disease table. These data sets are required for the operation of the model, and the values must appear on the punched cards as specified. The description of each input data set includes:

2) A general description--one or two sentences describing the data set and the information that it contains.
3) The name of each data set element--six or less alphanumeric characters used to designate the particular data set element.
4) The index, where applicable, indicated in parentheses immediately following the data set element name.
5) The definition of each data set element.
6) The positions (card columns) on the punch card of each data set element.
7) The field specification (in Fortran notation) of each data set element on the punched card.

Input Data Set Number III-1

Data Set Name: Control Card

Submodel Using Data Set: Disease and Chronic Conditions Submodel

General Description: This card contains coefficients applied by the respective parameters for each of the diseases considered.

Data Set Elements: These parameters indicate changes in equilibrium conditions because of fallout, etc.

Name	Definition	Card Column	Format
IPØPB	Population of area before attack.	1-10	I10
IPØPI	Population of area immediately after attack.	11-20	I10
IPØPA	Population of area remaining 30 days attack. (used in disease submodel)	21-30	I10
ILØC (1-5)	Area name or identification for disease submodel.	31-50	5A4
CAL	Coefficient of infective ratio such that the thus adjusted infective ratio AL' is: $AL' = 1 - \frac{1-AL}{CAL}$ where AL is the original infective ratio from the disease table. CAL increases with the radiation dose. It can assume any positive real value.	51-55	F5.2
CTB	Coefficient of contact rate such that the adjusted contact rate TB' is: TB' = CTB x TB; where TB is the original contact rate from the disease table. CTB increases with inadequacy of hygiene and sanitation measures and decreases with its adequacy. It can assume any positive real value.	56-60	F5.2
CSSS	Coefficient of initial infective fraction such that the thus adjusted initial infective fraction SSS' is: SSS' = CSS x SSS where SSS is the original susceptible fraction from the disease table. CSSS decreases with the efficiency of quarantine. It can assume any positive real value under 1, including 0.	61-65	F5.2

Name	Definition	Card Column	Format
CSR	Coefficient of initial susceptible fraction such that the thus adjusted initial susceptible fraction SR' is: $SR' = 1 - \frac{1-SR}{CSR}$; where SR is the original susceptible fraction from the disease table. CSR decreases with the adequacy of the preattack vaccination status and with the postattack level of antibiotic prophylaxis. CSR can assume any positive real value.	66-70	F5.2

Input Data Set Number III-2

Data Set Name: Disease Table

Submodel Using Data Set: Disease and Chronic Conditions Submodel

General Description: This card contains, for each one of the considered diseases separately, the parameters as listed below.

Data Set Elements:

Name	Definition	Card Column	Format
DIN	Duration of infectivity (in days)	1-10	F10.5
TB	Contact rate (per day)	11-20	F10.5
AL	Infective ratio	21-30	F10.5
SS	Fatality ratio (per new case)	31-40	F10.5
SR	Initial susceptible fraction	41-50	F10.5
SSS	Initial infective fraction	51-60	F10.5

IV. OUTPUTS FROM THE IMMEDIATE EFFECTS SUBMODEL

This subsection contains descriptions of the outputs from the Immediate Effects Submodel. Included in the description of each display data set are the following:

1) The data set name.
2) The submodel(s) which generate the output contained in the specified output data set.
3) A general description--one or two sentences describing the data set and the information it contains.
4) The name of each data set element--six or less alphanumeric characters used to designate the particular data set element. Where applicable, the appropriate index is designated in parentheses immediately following the data set element name.
5) The definition of each data set element--a concise definition of the data set element including its units.
6) A sample printout of the data set.

The remainder of this section is devoted to the presentation of the output data sets.

Output Data Set Number IV-1

Data Set Name: Casualty Treatment Table

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table displays the results of the triage of casualties assigned to each emergency treatment center.

The number of casualties assigned to each of the four treatment levels are totaled by column under the appropriate treatment level that appears at the top of the printout; i.e., No Treatment, Surgical Team, Physician Personnel, and Allied Medical Personnel. The heading "No Treatment" is used to indicate Treatment Level 1; i.e., the category of casualties due to receive no treatment at all, either because there is little or no prospect of recovery or because the injury is so slight as not to require treatment. The last three treatment levels (excluding the "no treatments") appear as headings for two columns--one for casualties which can be downgraded and another for those which cannot.

Each row shows the level of treatment that was actually administered. For example, the row total for Allied Medical Personnel (row heading appears down left side of printout) indicates that 32572 casualties were treated by Allied Medical Personnel--26860 that were assigned to be treated at this level and 5712 that were assigned treatment by Physician Personnel but were downgraded to the Allied Medical Personnel level.

As another example, the column total under the "Physician Personnel" heading indicates that 10425 casualties were assigned treatment at this level. However, only 4713 were actually treated by Physician Personnel--5712 were given downgraded treatment by Allied Medical Personnel.

Data Set Elements:

Name	Definition
NS (I)	Number of casualties treated by surgical teams
NP (I)	Number of casualties treated by physicians
NA (I)	Number of casualties treated by allied medical personnel
NT (I)*	Number of casualties transferred to the hospital for treatment
NU (I)	Number of casualties not treated
MTØT (I)	Total number of casualties processed

* Omitted from Table for the Hospital Emergency Treatment Center and for casualties transferred from other Emergency Treatment Centers.

Sample Output Showing Format Specifications:

NUMBER OF CASUALTIES AT EACH ASSIGNED LEVEL OF TREATMENT

(All Values are in Tens)

LEVEL AT WHICH TREATMENT WAS GIVEN	NO TREATMENT	TREATMENT LEVEL MAY BE DOWNGRADED			TREATMENT LEVEL MAY NOT BE DOWNGRADED			TOTAL
		SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	
SURGICAL TEAM	0	36	0	0	0	0	0	36
PHYSICIAN PERSONNEL	0	0	4713	0	0	0	0	4713
ALLIED MEDICAL PERSONNEL	0	0	5712	26860	0	0	0	32572
UNTREATED	1382	0	0	0	0	0	0	1382
TOTAL	1382	36	10429	26860	0	0	0	38703

Output Data Set Number IV-2

Data Set Name: Status of Supply Inventory

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table displays the initial and final inventory figures for the quantity consumed in processing the casualties for each of the sixteen medical treatment packages. The headings of the inventory table columns are as follows:

BI	Burn, non-ambulatory	LF	Fracture of leg
A	Abdominal	W	Laceration
T	Thoracic	H	Head
P	Pelvis, genito-urinary	HF	Fracture of hand
S	Shock	FF	Fracture of foot
M	Maxillofacial	E	Eye
AF	Fracture of arm	V	Vertebrae
BA	Burn, ambulatory		

Data Set Elements:

Name	Definition
NPACK (1,J)	The initial inventory, before processing the casualties
NPACK (2,J)	The final inventory, after processing the casualties
NPACK (3,J)	The amount used from the inventory to process the casualties

Sample Output Showing Format Specifications:

STATUS OF INVENTORY OF EACH MEDICAL TREATMENT PACKAGE

(All Values are in Tens)

	BI	A	T	P	S	M	AF	BA
INITIAL	181	23	167	183	179	1000	0	10050
FINAL	44	23	165	183	32	901	0	13771
USED	97	0	2	0	147	499	0	3187

	LF	W	H	HF	FF	E	V
INITIAL	0	349	37244	0	0	0	188
FINAL	0	198	32687	0	0	0	184
USED	0	234	283	0	0	0	0

Output Data Set Number IV-3

Data Set Name: Casualty Deaths Table

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table displays the results of processing the casualties assigned to each Emergency Treatment Center. It shows the number of deaths at each level of treatment, broken down by assigned treatment level and downgrading category, corresponding in form to the table described for Output Data Set IV-1.

Data Set Elements:

Name	Definition
NS(I + 10)	Number of deaths among casualties treated by surgical teams
NP(I + 10)	Number of deaths among casualties treated by physicians
NA(I + 10)	Number of deaths among casualties treated by allied medical personnel
NU(I + 10)	Number of deaths among untreated casualties
MTØT(I + 10)	Total number of deaths among all casualties

Sample Output Showing Format Specifications:

NUMBER OF DEATHS AT EACH ASSIGNED TREATMENT LEVEL

(All Values are in Tens)

LEVEL AT WHICH TREATMENT WAS GIVEN	NO TREATMENT	TREATMENT LEVEL MAY BE DOWNGRADED			TREATMENT LEVEL MAY NOT BE DOWNGRADED			TOTAL
		SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	
SURGICAL TEAM	0	10	0	0	0	0	0	10
PHYSICIAN PERSONNEL	0	0	960	0	0	0	0	960
ALLIED MEDICAL PERSONNEL	0	0	170	10790	0	0	0	10960
UNTREATED	960	0	0	0	0	0	0	960
TOTAL	960	10	1130	10790	0	0	0	10890

Output Data Set Number IV-4

Data Set Name: Personnel Utilization Table

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table gives the number of personnel assigned to the Emergency Treatment Center and the total number of hours these personnel spent on duty to attend to the load of casualties received by the center or hospital.

Data Set Elements:

Name	Description
NSRGT	Number of surgical teams assigned to the hospital
TSRGT	Number of hours surgical teams have been on duty
NPHYS	Number of physicians assigned to the Emergency Treatment Center
TPHYS	Number of hours physicians have been on duty
NALLD	Number of allied medical personnel assigned to the Emergency Treatment Center
TALLD	Number of hours allied medical personnel have been on duty

Sample Output Showing Format Specifications:

	NUMBER OF ASSIGNED PERSONNEL	TIME CLOCK STATUS AFTER TREATMENT
SURGICAL TEAM	40	30.04
PHYSICIAN PERSONNEL	18	30.03
ALLIED MEDICAL PERSONNEL	400	2.34
TOTAL		

Output Data Set Number IV-5

Data Set Name: Casualties Lacking Supplies Table

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table indicates the disposition of those casualties for whom medical treatment packages were lacking, including whether or not they were transferred to the hospital.

Data Set Elements:

Name	Description
KØWM(1,I)	Number of casualties lacking supplies at surgical team level
KØWM(2,I)	Number of casualties lacking supplies at physician levels
KØWM(3,I)	Number of casualties lacking supplies at allied medical personnel level
KØWM(4,I)	Number of all casualties lacking supplies

Sample Output Showing Format Specifications:

CASUALTIES WITHOUT MEDICAL SUPPLIES

	TRANSFERRED TO HOSPITAL	NOT TRANSFERRED	TOTAL
SURGICAL TEAM	0	628	628
PHYSICIAN PERSONNEL	0	332	332
ALLIED MEDICAL PERSONNEL	0	0	0
TOTAL	0	960	960

Output Data Set Number IV-6

Data Set Name: Casualty Treatment Table - Grand Total

Submodel Using Data Set: Immediate Effects Submodel

General Description: Same as Output Data Set IV-1, except that the data are totals of all Emergency Treatment Centers and the hospital.

Data Set Elements:

Name	Definition
NS(I+30)	Total number of casualties treated by surgical teams
NP(I+30)	Total number of casualties treated by physicians
NA(I+30)	Total number of casualties treated by allied medical personnel
NT(I+30)	Total number of casualties transferred from Emergency Treatment Centers to hospitals
NU(I+30)	Total number of untreated casualties
MTØT(I+30)	Total number of casualties

Sample Output Showing Format Specifications:

GRAND TOTALS. LOWELL, MARKED CONSERVATIVE, M, PERSONNEL DOUBLED

LEVEL AT WHICH TREATMENT WAS GIVEN	NUMBER OF CASUALTIES AT EACH ASSIGNED LEVEL OF TREATMENT							
	EXPECTANT OF TREATMENT	TREATMENT LEVEL MAY BE DOWNGRADED			TREATMENT LEVEL MAY NOT BE DOWNGRADED			
		SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	TOTAL
SURGICAL TEAM	0	98*	0	0	0	0	0	980
PHYSICIAN PERSONNEL	0	714	031	2	0	0	0	047
ALLIED MEDICAL PERSONNEL	0	*	1854	5430	0	0	0	7404
TRANSFERS TO HOSPITAL GAIN	0	5214	01	1009	0	0	0	0327
UNTREATED	93.00	5914	0	0	0	0	0	98724
TOTAL	93260	611*	2403	5032	0	0	0	8*030

Output Data Set Number IV-7

Data Set Name: Status of Supply Inventory - Grand Total

Submodel Using Data Set: Immediate Effects Submodel

General Description: Same as Output Data Set IV-2 except that the data are totals of all Emergency Treatment Centers and the hospital.

Data Set Elements:

Name	Definition
NPACK(4,J)	The total initial inventory, before processing the casualties
NPACK(5,J)	The total final inventory, after processing the casualties
NPACK(6,J)	The total amount used from the inventory to process the casualties

Sample Output Showing Format Specifications:

STATUS OF INVENTORY OF EACH MEDICAL TREATMENT PACKAGE	BI	A	T	P	S	M	AF	RA
INITIAL	1793	121	121	141	670	151	179982	1884
FINAL	1792	91	91	91	670	151	179982	1884
USED	1	30	30	50	0	0	0	0

	LF	W	M	WF	FF	E	V
INITIAL	179830	147	552	179982	179444	481	179931
FINAL	177724	147	0	179982	179616	481	179823
USED	1936	0	552	0	2828	0	81

Output Data Set Number IV-8

Data Set Name: Casualty Deaths Table - Grand Total

Submodel Using Data Set: Immediate Effects Submodel

General Description: Same as Output Data Set IV-3 except that the data are totals of all Emergency Treatment Centers and the hospital.

Data Set Elements:

Name	Definition
NS(I+40)	Total number of deaths among casualties treated by surgical teams
NP(I+40)	Total number of deaths among casualties treated by physicians
NA(I+40)	Total number of deaths among casualties treated by allied medical personnel
NU(I+40)	Total number of deaths among untreated casualties
MTØT(I+40)	Total number of deaths among all casualties

Sample Output Showing Format Specifications:

LEVEL AT WHICH TREATMENT WAS GIVEN	NUMBER OF EXPECTANT OF TREATMENT	DEATHS AT TREATMENT LEVEL MAY BE DOWNGRADED: SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	ASSIGNED TREATMENT LEVEL MAY NOT BE DOWNGRADED: SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	TOTAL
SURGICAL TEAM	0	443	0	0	0	0	0	443
PHYSICIAN PERSONNEL	0	191	359	0	0	0	0	550
ALLIED MEDICAL PERSONNEL	0	0	1151	1296	0	0	0	2447
UNTREATED	11744	4657	0	0	0	0	0	16401
TOTAL	11744	5291	1510	1296	0	0	0	19841

Output Data Set Number IV-9

Data Set Name: Personnel Utilization - Grand Total

Submodels Using Data Set: Immediate Effects Submodel

General Description: This table gives the total number of personnel assigned to the Emergency Treatment Centers and the hospital.

Data Set Elements:

Name	Definition
NNSRGT	Total number of surgical teams assigned to the hospital
NNPHYS	Total number of physicians assigned to the Emergency Treatment Center or hospitals
NNALLD	Total number of allied medical personnel assigned to the Emergency Treatment Center or hospitals

Sample Output Showing Format Specifications:

	NUMBER OF ASSIGNED PERSONNEL
SURGICAL TEAM	80
PHYSICIAN PERSONNEL	64
ALLIED MEDICAL PERSONNEL	1160

Output Data Set Number IV-10

Data Set Name: Casualties Lacking Supplies - Grand Total

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table indicates the disposition of those casualties for whom medical treatment packages were lacking; including whether or not they were transferred to the hospital.

Data Set Elements:

Name	Description
KØWM(5,I)	Total number of casualties lacking supplies at surgical team level
KØWM(6,I)	Total number of casualties lacking supplies at physician level
KØWM(7,I)	Total number of casualties lacking supplies at allied medical personnel level
KØWM(8,I)	Grand total of all casualties lacking supplies

Sample Output Showing Format Specifications:

	CASUALTIES WITHOUT MEDICAL SUPPLIES		
	TRANSFERRED TO HOSPITAL	NOT TRANSFERRED	T O T A L
SURGICAL TEAM	0	4282	4282
PHYSICIAN PERSONNEL	0	1964	1964
ALLIED MEDICAL PERSONNEL	0	930	930
TOTAL	0	7176	7176

Output Data Set Number IV-11

Data Set Name: Table of Survivors Added - Grand Total

Submodel Using Data Set: Immediate Effects Submodel

General Description: This table displays the total number of survivors which resulted from changes in personnel and medical supply inventories above the number found in the base run. The number added survivors will be negative when the change results in fewer survivors.

Data Set Elements:

Name	Description
NS(I+50)	Total number of added survivors treated by surgical teams
NP(I+50)	Total number of added survivors treated by physicians
NA(I+50)	Total number of added survivors treated by allied medical personnel
NU(I+50)	Total number of added survivors untreated
MTØT(I+50)	Grand total of survivors added

Sample Output Showing Format Specifications:

LEVEL AT WHICH TREATMENT WAS GIVEN	NUMBER OF EXPECTANT OF TREATMENT	SURVIVORS ADDED AT TREATMENT LEVEL MAY BE DOWNGRADED TO SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	SURVIVORS ADDED AT EACH TREATMENT LEVEL MAY NOT BE DOWNGRADED TO SURGICAL TEAM	PHYSICIAN PERSONNEL	ALLIED MEDICAL PERSONNEL	TOTAL
SURGICAL TEAM	0	-254	0	0	0	0	0	-254
PHYSICIAN PERSONNEL	0	75	-359	0	0	0	0	-284
ALLIED MEDICAL PERSONNEL	0	0	3287	-840	0	0	0	2447
UNTREATED	-5975	9451	0	0	0	0	0	3476
TOTAL	-5975	9272	2928	-840	0	0	0	5385

V. OUTPUTS FROM DISEASE AND CHRONIC CONDITIONS SUBMODEL

This subsection contains a description of the four outputs from the Disease and Chronic Conditions Submodel, i.e., Plot of Infectives and Required Physicians, New Infective Table, Death Table, and Drug Requirement Table. The description of each data set are the following:

1) The data set name.
2) A general description--one or two sentences describing the data set and and the information it contains.
3) The name of each data set element--six or less alphanumeric characters used to designate the particular data set element. Where applicable, the appropriate index is designated in parentheses immediately following the data set element name.
4) The definition of each data set element--a concise definition of the data set element including its units.
5) A sample printout of the data set.

Output Data Set Number V-I

Data Set Name: New Infective Table

Submodel Using Data Set: Disease and Chronic Conditions Submodel

General Description: A table displaying the total number of new infectives expressed as a fraction of the total population, by five-day periods and by disease.

Data Set Elements:

Name	Definition
RIP	Fraction of population that became infected during time interval DT.

Sample Output Showing Format Specifications:

NUMBER OF NEW INFECTIVES
BY DISEASE AND BY 5-DAY PERIOD

LOCATION OF ATTACK- NEW ORLEANS - MAX S
RES POP BEFORE ATTACK- 1002000
RES POP IMM AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 817000

LAST DAY IN PERIOD	DIPH	DISE	GAST	DISEASE HEPA	INFL	MEAS	PARA	SCAR	WHOO	TOTAL
5	180.	140.	384.	180.	172.	139.	180.	180.	155.	1749.
10	262.	229.	2517.	245.	270.	204.	212.	262.	163.	4364.
15	392.	311.	17243.	360.	417.	294.	262.	384.	172.	19833.
20	564.	417.	49557.	515.	621.	425.	335.	564.	180.	93176.
25	817.	558.	108821.	719.	915.	588.	417.	809.	188.	185829.
30	1177.	744.	118216.	909.	1324.	793.	523.	1152.	204.	125120.
35	1659.	997.	48149.	1340.	1904.	1045.	662.	1618.	221.	57596.
40	2394.	1342.	17476.	1757.	2705.	1316.	825.	2223.	237.	30375.
45	3114.	1785.	6301.	2247.	3800.	1594.	1030.	2975.	253.	23077.
50	4078.	2337.	2198.	2754.	5263.	1822.	1291.	3857.	270.	23870.
55	5132.	3041.	735.	3228.	7175.	1978.	1610.	4789.	294.	26022.
60	6137.	4047.	212.	3596.	9561.	2035.	2002.	5547.	311.	33538.
65	6934.	5255.	53.	3800.	12397.	1986.	2484.	6301.	327.	39519.
70	7387.	6748.	0.	3824.	15527.	1847.	3073.	6619.	343.	45387.
75	7429.	8605.	0.	3661.	18640.	1659.	3775.	6578.	368.	50707.
80	7069.	10771.	0.	3374.	21321.	1438.	4617.	6219.	384.	55193.
85	6439.	13222.	0.	3007.	23135.	1225.	5614.	5630.	409.	58683.
90	5695.	15845.	0.	2607.	23780.	1021.	6766.	4928.	425.	61034.
95	4938.	18469.	0.	2215.	23208.	842.	8066.	4200.	449.	62286.
100	4037.	20863.	0.	1855.	21590.	686.	9520.	3506.	466.	62524.
105	3320.	22783.	0.	1536.	19286.	556.	11073.	2885.	482.	61927.
110	2705.	24044.	0.	1258.	16646.	449.	12491.	2345.	507.	60634.
115	2174.	24485.	0.	1021.	13974.	364.	14285.	1896.	523.	58723.
120	1741.	24140.	0.	834.	11482.	294.	15788.	1520.	539.	56337.
125	1385.	23110.	0.	670.	9275.	245.	17096.	1218.	556.	53 59.
130	1104.	21588.	0.	548.	7404.	196.	18117.	972.	572.	50470.
135	874.	19678.	0.	441.	5851.	163.	18795.	768.	588.	47160.
140	695.	17643.	0.	360.	4584.	139.	19098.	613.	597.	43728.
145	548.	15542.	0.	294.	3579.	114.	19016.	490.	613.	40247.
150	438.	13615.	0.	237.	2787.	98.	18575.	384.	621.	36749.
155	348.	11774.	0.	196.	2157.	82.	17848.	311.	629.	33341.
160	278.	10179.	0.	163.	1675.	74.	16883.	245.	637.	30054.
165	212.	8821.	0.	131.	1299.	65.	15764.	196.	637.	26927.
170	172.	7592.	0.	114.	1005.	57.	14554.	155.	646.	24024.
175	139.	6534.	0.	90.	785.	49.	13304.	131.	646.	21337.
180	114.	5622.	0.	82.	613.	49.	12062.	106.	646.	18893.
185	90.	4844.	0.	65.	482.	49.	10852.	82.	646.	16654.
190	74.	4188.	0.	57.	378.	41.	9716.	65.	637.	14652.
195	57.	3649.	0.	57.	302.	41.	8654.	57.	637.	12895.
200	49.	3202.	0.	49.	245.	41.	7673.	49.	629.	11318.
205	41.	2837.	0.	41.	198.	41.	6783.	41.	621.	9921.
210	33.	2538.	0.	41.	163.	41.	5974.	33.	613.	8695.
215	33.	2295.	0.	41.	131.	41.	5255.	33.	605.	7633.
220	25.	2100.	0.	33.	114.	41.	4609.	25.	597.	6693.
225	25.	1938.	0.	33.	98.	33.	4037.	25.	588.	5878.
230	16.	1806.	0.	33.	98.	33.	3522.	25.	572.	5154.
235	16.	1719.	0.	33.	82.	33.	3081.	16.	564.	4544.
240	16.	1637.	0.	33.	74.	33.	2680.	16.	548.	3996.
245	16.	1498.	0.	33.	65.	33.	2337.	16.	539.	3538.
250	16.	1419.	0.	25.	65.	33.	2035.	16.	523.	3122.
255	16.	1343.	0.	25.	57.	33.	1773.	16.	507.	2770.
260	8.	1278.	0.	25.	57.	41.	1536.	16.	498.	2460.
265	8.	1229.	0.	25.	57.	41.	1340.	16.	482.	2198.
270	8.	1188.	0.	25.	49.	41.	1180.	16.	466.	1953.
275	8.	1155.	0.	25.	49.	41.	1005.	16.	449.	1749.
280	8.	1131.	0.	25.	49.	41.	874.	16.	441.	1586.
285	8.	1106.	0.	25.	49.	41.	760.	16.	429.	1430.
290	8.	1082.	0.	25.	49.	41.	694.	16.	409.	1283.
295	8.	1065.	0.	25.	49.	41.	572.	16.	400.	1177.
300	8.	1057.	0.	25.	49.	41.	490.	16.	384.	1071.
305	8.	1041.	0.	25.	49.	41.	425.	16.	376.	981.
310	8.	1033.	0.	25.	49.	41.	368.	16.	360.	899.
315	8.	1025.	0.	25.	49.	41.	319.	16.	351.	834.
320	8.	1016.	0.	25.	49.	41.	278.	16.	335.	768.
325	8.	1008.	0.	25.	49.	41.	237.	16.	327.	719.
330	8.	992.	0.	25.	49.	41.	204.	16.	311.	662.
335	8.	984.	0.	33.	49.	41.	180.	16.	302.	637.
340	8.	976.	0.	33.	49.	41.	155.	16.	294.	597.
345	8.	968.	0.	33.	49.	41.	131.	16.	286.	564.
350	8.	960.	0.	33.	49.	41.	114.	16.	270.	531.
355	8.	951.	0.	33.	49.	41.	98.	16.	262.	507.
360	8.	943.	0.	33.	49.	41.	82.	16.	253.	482.
365	8.	935.	0.	33.	49.	41.	65.	16.	245.	458.
TOTAL	92934.	397995.	489040.	51439.	294249.	27399.	398222.	83544.	32808.	1868896.

Output Data Set Number V-2

Data Set Name: Fatality Table

Submodel Using Data Set: Disease and Chronic Conditions Submodel

General Description: This table displays the total number of fatalities expressed as a fraction of the initial population by five day periods and by disease.

Data Set Elements:

Name	Definition
DRD	Fraction of population that died during time interval DT.

Sample Output Showing Format Specifications:

NUMBER OF DEATHS
BY DISEASE AND BY 5-DAY PERIOD

LOCATION OF ATTACK- NEW ORLEANS - MAX 5
RES POP BEFORE ATTACK- 1002000
RES POP IMM AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 817000

LAST DAY IN PERIOD	DIPH	DISE	DISEASE GAST	MEPA	INFL	MEAS	PARA	SCAR	WHOO	TOTAL
5	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
10	0.	0.	25.	0.	0.	0.	0.	0.	0.	25.
15	0.	0.	189.	0.	0.	0.	0.	0.	0.	196.
20	0.	0.	997.	0.	0.	0.	0.	0.	0.	1021.
25	0.	0.	2010.	0.	0.	0.	0.	0.	0.	2035.
30	10.	0.	1316.	0.	0.	0.	0.	0.	0.	1348.
35	10.	0.	531.	0.	16.	0.	0.	0.	0.	588.
40	25.	10.	196.	0.	16.	0.	0.	0.	0.	270.
45	33.	16.	74.	0.	25.	0.	0.	0.	0.	163.
50	41.	25.	25.	0.	41.	0.	0.	0.	0.	155.
55	57.	33.	0.	0.	49.	0.	0.	0.	0.	172.
60	65.	41.	0.	0.	65.	0.	16.	0.	0.	204.
65	74.	49.	0.	16.	90.	0.	16.	0.	0.	253.
70	82.	65.	0.	16.	114.	0.	25.	0.	0.	311.
75	82.	90.	0.	0.	131.	0.	25.	0.	0.	335.
80	82.	106.	0.	0.	155.	0.	33.	0.	0.	384.
85	74.	131.	0.	0.	163.	0.	41.	0.	0.	417.
90	65.	155.	0.	0.	172.	0.	49.	0.	0.	449.
95	57.	188.	0.	0.	163.	0.	57.	0.	0.	474.
100	41.	212.	0.	0.	155.	0.	65.	0.	0.	482.
105	33.	229.	0.	0.	139.	0.	74.	0.	0.	482.
110	33.	247.	0.	0.	123.	0.	82.	0.	0.	482.
115	25.	245.	0.	0.	98.	0.	98.	0.	0.	466.
120	16.	245.	0.	0.	82.	0.	106.	0.	0.	449.
125	10.	229.	0.	0.	65.	0.	114.	0.	0.	425.
130	0.	212.	0.	0.	49.	0.	123.	0.	0.	404.
135	0.	196.	0.	0.	41.	0.	123.	0.	0.	376.
140	0.	180.	0.	0.	33.	0.	131.	0.	0.	360.
145	0.	155.	0.	0.	25.	0.	131.	0.	0.	327.
150	0.	139.	0.	0.	16.	0.	123.	0.	0.	294.
155	0.	114.	0.	0.	16.	0.	123.	0.	0.	262.
160	0.	98.	0.	0.	0.	0.	114.	0.	0.	220.
165	0.	90.	0.	0.	0.	0.	106.	0.	0.	212.
170	0.	74.	0.	0.	0.	0.	98.	0.	0.	188.
175	0.	65.	0.	0.	0.	0.	90.	0.	0.	172.
180	0.	49.	0.	0.	0.	0.	82.	0.	0.	147.
185	0.	41.	0.	0.	0.	0.	74.	0.	0.	123.
190	0.	41.	0.	0.	0.	0.	65.	0.	0.	114.
195	0.	33.	0.	0.	0.	0.	57.	0.	0.	98.
200	0.	25.	0.	0.	0.	0.	49.	0.	0.	82.
205	0.	25.	0.	0.	0.	0.	49.	0.	0.	82.
210	0.	16.	0.	0.	0.	0.	41.	0.	0.	65.
215	0.	16.	0.	0.	0.	0.	33.	0.	0.	57.
220	0.	16.	0.	0.	0.	0.	33.	0.	0.	57.
225	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
230	0.	0.	0.	0.	0.	0.	25.	0.	0.	41.
235	0.	0.	0.	0.	0.	0.	25.	0.	0.	33.
240	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
245	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
250	0.	0.	0.	0.	0.	0.	16.	0.	0.	25.
255	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
260	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
265	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
270	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
275	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
280	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
285	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
290	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
295	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
300	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
305	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
310	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
315	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
320	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
325	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
330	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
335	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
340	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
345	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
350	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
355	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
360	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
365	0.	0.	0.	0.	0.	0.	0.	0.	0.	0.
TOTAL	997.	3940.	9377.	147.	2188.	41.	2872.	0.	172.	19494.

Output Data Set Number V-3

Data Set Name: Plot of Infectives and Required Physicians

Submodel Using Data Set: Disease and Chronic Conditions Submodel

General Description: This output consists of a list and graphic plot of the total number of infectives and the corresponding physician requirement for every fifth day during the 1 year postattack period.

Data Set Elements:

Name	Definition
RSTØ	Total
TPT	Number of Physicians Required

Sample Output Showing Format Specifications:

PLOT OF INFECTIVES AND REQ PHYSICIANS

LOCATION OF ATTACK- NEW ORLEANS - MAX 9
RES POP BEFORE ATTACK- 1002000
RES POP 1HR AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 817000

PHY AVAILABLE(12HR/DAY)- 1301

DAYS	NUMBER OF INFECTIVES	PHYSICIANS REQUIRED FOR DISEASE TREATMENT (12HR / PHYSICIAN DAY)
5	3807.	48.
10	6305.	84.
15	19397.	259.
20	75282.	1003.
25	120650.	1009.
30	72981.	972.
35	[illegible]	540.
40	[illegible]	420.
45	[illegible]	451.
50	[illegible]	543.
55	[illegible]	671.
60	[illegible]	828.
65	7344	979.
70	[illegible]	1138.
75	[illegible]	1298.
[illegible]	[illegible]	1424.
[illegible]	[illegible]	1545.
90	[illegible]	1658.
95	[illegible]	1748.
100	[illegible]	1816.
105	[illegible]	1868.
110	[illegible]	1938.
115	[illegible]	1963.
[illegible]	[illegible]	1976.
[illegible]	[illegible]	1968.
[illegible]	[illegible]	1938.
[illegible]	[illegible]	1868.
[illegible]	[illegible]	1828.
[illegible]	[illegible]	1737.
[illegible]	[illegible]	1642.
[illegible]	[illegible]	1537.
[illegible]	[illegible]	1427.
[illegible]	[illegible]	1314.
[illegible]	[illegible]	1201.
[illegible]	[illegible]	1092.
[illegible]	[illegible]	987
[illegible]	[illegible]	889.
[illegible]	[illegible]	797.
[illegible]	[illegible]	714.
[illegible]	[illegible]	638.
[illegible]	[illegible]	559.
[illegible]	[illegible]	508.
[illegible]	[illegible]	453.
[illegible]	[illegible]	425.
[illegible]	[illegible]	352.
[illegible]	[illegible]	324
[illegible]	[illegible]	281
[illegible]	[illegible]	262.
[illegible]	[illegible]	238
[illegible]	[illegible]	214.
[illegible]	[illegible]	188.
[illegible]	[illegible]	170.
[illegible]	[illegible]	151.
[illegible]	[illegible]	147
[illegible]	[illegible]	135.
[illegible]	[illegible]	124.
[illegible]	[illegible]	115
[illegible]	[illegible]	120.
[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	92.
[illegible]	[illegible]	88
[illegible]	[illegible]	81
[illegible]	[illegible]	76
[illegible]	[illegible]	76.
[illegible]	[illegible]	68
[illegible]	[illegible]	64
[illegible]	[illegible]	61.
[illegible]	[illegible]	58
[illegible]	[illegible]	55
[illegible]	[illegible]	52.
[illegible]	[illegible]	58
[illegible]	[illegible]	48.
[illegible]	[illegible]	48

[illegible]	DURATION	CONTACT RATE	SUSCEPTIBLE RATIO	INFECTIVE RATIO	DEATH RATE	INITIAL INFECTION
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

Output Data Set Number V-4

Data Set Name: Drug Requirements

Submodel Using Data Set: Disease and Chronic Conditions Submodel

General Description: This table displays the five day and cumulative drug requirements for the 1 year postattack period.

Data Set Element:

Name		Definition
(5 day name)	(total name)	
DRUA	DRTA	Intravenous infusion required
DRUB	DRTB	Penicillin units required
DRUC	DRTC	Broad-spectrum antibiotics required
DRUD	DRTD	Streptomycin required

Sample Output Showing Format Specifications:

DRUG REQUIREMENTS - BY 5 DAY PERIOD AND CUMULATIVE TOTAL

LOCATION OF ATTACK- NEW ORLEANS - MAX S
RES POP BEFORE ATTACK- 1002000
RES POP IMM AFTER ATTACK- 1000000
POP 30 DAYS AFTER ATTACK- 817000

INT INFUSION AVAILABLE- 57000 UNITS
PENICILLIN AVAILABLE- 281000 M UNITS
B-S ANTIBIO AVAILABLE- 1750000 GRAMS
STREPTOMYCIN AVAILABLE- 48000 GRAMS

	INTRAVENOUS INFUSION UNITS		PENICILLIN OR SULFA MILLIONS OF UNITS		BROAD-SPECT ANTIBIO GRAMS		STREPTOMYCIN GRAMS	
DAY	USED	TOT USED	USED	TOT USED	USED	TOT USED	USED	TOT USED
5	33617.	33617.	1054.	1054.	2855.	2859.	2788.	2788.
10	125702.	159318.	1610.	2664.	7709.	10564.	2940.	5728.
15	723254.	882572.	2356.	5020.	37961.	48525.	3113.	8841.
20	3437824.	4320384.	3437.	8458.	174186.	222711.	3311.	12153.
25	5597648.	9918016.	4945.	13402.	282750.	505461.	3538.	15691.
30	3060424.	12978430.	7029.	20431.	156628.	662088.	3796.	19487.
35	1267684.	14246119.	9853.	30284.	67920.	730008.	4091.	23577.
40	566616.	14812703.	13581.	43865.	34016.	764022.	4427.	28004.
45	343941.	15156838.	18336.	62201.	24322.	788342.	4809.	32813.
50	310696.	15467327.	24140.	86340.	24452.	812794.	5191.	38004.
55	359817.	15827134.	30842.	117181.	29131.	841924.	5529.	43533.
60	456111.	16283230.	38065.	155246.	36696.	878620.	5890.	49422.
65	591570.	16874750.	45213.	200459.	46857.	925476.	6273.	55695.
70	767224.	17641918.	51562.	252021.	59789.	985264.	6678.	62373.
75	984524.	18626431.	56410.	308430.	75702.	1060964.	7102.	69474.
80	1246480.	19872895.	59219.	367642.	94889.	1155852.	7543.	77017.
85	1551912.	21424766.	59704.	427351.	117427.	1273276.	7997.	85014.
90	1893200.	23317950.	57869.	485222.	143054.	1416328.	8459.	93473.
95	2254350.	25572286.	54021.	539242.	171042.	1587368.	8923.	102396.
100	2610744.	28182975.	48703.	587944.	200156.	1787524.	9387.	111783.
105	2931400.	31114366.	42572.	630516.	228738.	2016260.	9856.	121639.
110	3184400.	34298750.	36244.	666760.	254937.	2271192.	10328.	131966.
115	3343800.	37642494.	30195.	696951.	277044.	2548232.	10799.	142765.
120	3395888.	41038334.	24725.	721678.	293793.	2842024.	11266.	154031.
125	3341800.	44380030.	19975.	741652.	304524.	3146552.	11724.	165755.
130	3195840.	47575805.	15975.	757626.	309190.	3455736.	12170.	177925.
135	2980584.	50554285.	12682.	770308.	308160.	3763896.	12600.	190525.
140	2721136.	53277310.	10014.	780322.	302084.	4065976.	13012.	203537.
145	2440520.	55717757.	7882.	788200.	291733.	4357696.	13401.	216938.
150	2157224.	57874981.	6192.	794392.	277923.	4635616.	13767.	230704.
155	1884448.	59759358.	4863.	799254.	261492.	4897088.	14105.	244809.
160	1630504.	61389821.	3823.	803076.	243223.	5140304.	14414.	259223.
165	1400128.	62789985.	3011.	806086.	223911.	5364208.	14690.	273913.
170	1194872.	63984638.	2381.	808464.	204230.	5568432.	14932.	288845.
175	1011836.	64990204.	1891.	810356.	184750.	5753184.	15138.	303983.
180	858184.	65857405.	1512.	811868.	165951.	5919120.	15307.	319289.
185	723440.	66580733.	1218.	813084.	148136.	6067248.	15438.	334726.
190	602250.	67148732.	991.	814074.	131513.	6198768.	15530.	350256.
195	510211.	67638941.	815.	814888.	116263.	6315024.	15585.	365841.
200	427318.	68112205.	580.	815568.	102370.	6417392.	15603.	381443.
205	357298.	68461375.	475.	816188.	89844.	6507232.	15583.	397026.
210	296305.	68741585.	443.	816632.	78634.	6585856.	15528.	412554.
215	248870.	68990398.	431.	817062.	68663.	6654512.	15441.	427994.
220	207375.	69217757.	382.	817444.	59830.	6714336.	15321.	443314.
225	172639.	69410301.	345.	817724.	52042.	6764384.	15171.	458484.
230	143583.	69553661.	318.	818[illegible]	49213.	6811616.	14993.	473477.
235	119171.	69672957.	294.	818394.	39252.	6850844.	14700.	488207.
240	98043.	69771772.	277.	818677.	34827.	6884000.	14569.	502831.
245	82130.	69853894.	263.	818933.	29871.	6914330.	14319.	517149.
250	68062.	69921532.	253.	819184.	25506.	6939840.	14055.	531202.
255	56258.	69977590.	245.	819429.	22050.	6961080.	13775.	544976.
260	46451.	70023932.	240.	819669.	19842.	6980944.	13483.	558459.
265	38275.	70062078.	235.	819904.	16440.	6997400.	13180.	571638.
270	31490.	70093109.	232.	820139.	14218.	7011610.	12887.	584502.
275	25700.	70119008.	229.	820358.	12260.	7023872.	12549.	597052.
280	21044.	70139998.	228.	820586.	10970.	7034448.	12225.	609274.
285	17170.	70156798.	226.	820814.	9117.	7043952.	11898.	621172.
290	14010.	70170305.	226.	821036.	7851.	7051392.	11578.	632742.
295	11068.	70181373.	225.	821259.	6797.	7058144.	11262.	643982.
300	8751.	70190070.	225.	821482.	5816.	7064992.	10915.	654896.
305	6874.	70196711.	225.	821706.	4900.	7069858.	10590.	665496.
310	5244.	70201954.	225.	821932.	4282.	7073280.	10240.	675754.
315	1957.	70205603.	225.	822154.	3640.	7076800.	9993.	689706.
320	2850.	70208518.	224.	822378.	3130.	7080000.	9441.	695294.
325	1020.	70210301.	226.	822604.	2680.	7082672.	9319.	704680.
330	1155.	70211329.	227.	822831.	2283.	7084944.	9036.	713716.
335	511.	70211592.	227.	823058.	1930.	7086808.	8743.	722456.
340	0.	70211592.	228.	823282.	1643.	7088512.	8456.	730912.
345	0.	70211592.	229.	823511.	1400.	7089920.	8186.	739098.
350	0.	70211592.	229.	823738.	1204.	7091124.	7910.	747008.
355	0.	70211592.	230.	823966.	1027.	7092144.	7648.	754644.
360	0.	70211592.	231.	824199.	873.	7093000.	7390.	762036.
365	0.	70211592.	231.	824424.	730.	7093760.	7140.	769182.

Appendix E

The Fifteen Leading Causes of Death in Selected Countries

This appendix lists the fifteen leading causes of death for two contemporary under-developed countries (Nigeria and Portugal), for the U.S. in 1900, and in comparison, for the U.S. in 1964. These data serve to amplify the Background Section in Chapter 3, Disease and Chronic Conditions Submodel and refers directly to Figure 6 in the main text.

Table E-1

FIFTEEN LEADING CAUSES OF DEATH IN NIGERIA, 1960 AND PORTUGAL, 1962

Disease Categories and ICD Code Numbers	NIGERIA[1/] 1960 % Total	NIGERIA[1/] 1960 Cum. %	PORTUGAL[1/] 1962	PORTUGAL[1/] 1962 % Total	PORTUGAL[1/] 1962 Cum. %
Pneumonia and Influenza (480-493)	15.47	15.47	Diseases of Heart (400-402,410-443)	15.02	15.02
Senility without mention of Psychosis (780-795)	14.61	30.08	Senility without mention of Psychosis (780-795)	13.25	28.27
Certain Diseases of Early Infancy (760-776)	11.60	41.68	Vascular Lesions Affecting Central Nervous System (330-334)	12.95	41.22
Malaria (110-117)	10.09	51.79	Malignant Neoplasms (140-205)	9.72	50.94
Gastritis (543, 571, 572)	8.45	60.22	Pneumonia and Influenza (480-493)	8.82	59.76
Infective and Parasitic (030-039,041,042,044,049,052-054, 059-074,081-083,086-096,120-138)	4.75	64.97	Gastritis (543, 571, 572)	7.69	67.45
Accidents (E 800-E 962)	4.26	69.23	Certain Diseases of Early Infancy (760-776)	6.57	74.02
Birth Injuries, Postnatal Asphyxia and Atelectasis (760-762)	3.59	72.82	Accidents (E 800-E 962)	3.79	77.81
Tuberculosis (all forms) (001-019)	2.84	75.66	Tuberculosis (all forms) (001-019)	3.40	81.21
Vascular Lesions affecting Central Nervous System (330-334)	2.35	78.01	Bronchitis (500-502)	2.89	84.10
Deliveries and Complications of Pregnancy (640-689)	1.77	79.78	Cirrhosis of Liver (581)	2.10	86.20
Dysentary (all forms) (045-048)	1.65	81.43	Nephritis and Nephrosis (590-594)	1.76	87.96
Non-Meningococcal Meningitis (340)	1.47	82.90	Ulcer of Stomach and Duodenum (540, 541)	.89	88.85
Anemias (290-293)	1.42	84.32	Suicide and Self-Inflicted Injury (E 963, E 970-E979)	.80	89.65
Bronchitis (500-502)	1.29	85.61	Diabetes Mellitus (260)	.72	90.37

Source:

1/ Demographic Yearbook 1963. New York: United Nations, p. 592, 1964. (The Sixth Revision of the International Classification of Diseases is used here.)

Table E-1 (Continued)

FIFTEEN LEADING CAUSES OF DEATH IN THE UNITED STATES, 1900 and 1964

Disease Categories and ICD Code Numbers	UNITED STATES[1/ 2/] 1900 % Total	UNITED STATES[1/ 2/] 1900 Cum. %	UNITED STATES[3/] 1964	UNITED STATES[3/] 1964 % Total	UNITED STATES[3/] 1964 Cum. %
Pneumonia (all forms) and Influenza (480-493)	11.76	11.76	Diseases of Heart (400-402,410-443)	38.9	38.9
Tuberculosis (all forms) (001-019)	11.31	23.07	Malignant Neoplasms (140-205)	16.1	55.0
Diarrhea, Enteritis and Ulceration of Intestine (543,571,572)	8.30	31.37	Vascular Lesions Affecting Central Nervous Systems (330-334)	11.0	66.0
Diseases of Heart (410-443)	7.99	39.36	Accidents (E 800-E 962)	5.8	71.8
Senility, Ill Defined or Unknown (780-795)	6.83	46.19	Certain Diseases of Infancy (760-776)	3.4	75.2
Intracranial Lesions of Vascular Organ (330-334)	6.22	52.41	Influenza and Pneumonia, except Pneumonia of Newborn (480-493)	3.3	78.5
Nephritis (590,591,592-594)	5.15	57.56	General Arteriosclerosis (450)	2.1	80.6
All Accidents (E 800-E 802, E 840-E 962)	4.21	61.77	Diabetes Mellitus (260)	1.8	82.4
Cancer and Other Malignant Tumors (140-205)	3.72	65.49	Other Diseases of Circulatory System (451-468)	1.4	83.8
Diphteria (055)	2.34	67.83	Other Bronchopulmonic Diseases (525-527)	1.3	85.1
Simple Meningitis* (61)	1.97	69.80	Cirrhosis of Liver (581)	1.3	86.4
Typhoid Fever (Abdominal Typhus) (040)	1.82	71.62	Suicide (E 963, E 970-E 979)	1.1	87.5
Acute Bronchitis* (90)	1.53	73.15	Congenital Malformation (750-759)	1.1	88.6
Paralysis without specified cause* (66)	1.52	74.67	Other Hypertensive Disease (444-447)	0.7	89.3
Convulsions* (under 5 years) (71)	1.33	76.00	Nephritis and Nephrosis (590-594)	0.7	90.0

Sources:

1/ Vital Statistics of the United States 1950. Washington, D. C.: U. S. Government Printing Office. Vol. I, Tables 8:45, p. 218. (The Sixth Revision of the International Classification of Diseases is used except for diseases with an (*) where the First Revision is used.)

2/ Linder, Forest E. and Robert D. Grove. Vital Statistics Rates in the United States 1900-1940. Washington, D. C.: U. S. Government Printing Office, pp. 210-215, 1943.

3/ Vital Statistics of the United States 1964. Washington, D. C.: U. S. Government Printing Office. Vol. I, Table 1-6, p. 1-6. (The Seventh Revision of the International Classification of Diseases is used here.)

Appendix F

Epidemiologic Features of Communicable Diseases

This appendix discusses some salient epidemiologic features of communicable diseases. It deals briefly with man-to-man transmission (Section I), with food- or water-to-man transmission (Section II), and with vector borne diseases (Section III).

Appendix F

Epidemiologic Features of Communicable Diseases

I. MAN-TO-MAN TRANSMISSION

A. Introduction

Most of the mathematical modeling of disease has been of those transmitted from man-to-man. Given one infective in the group, the course of the disease spread depends upon several factors, the three most important of which are:

1) Immune status of the group members.
2) The likelihood that a susceptible will contract the disease if he comes in contact with an infective (called communicability, or infectiousness).
3) Mixing parameters which reflect the degree of contact among susceptibles and infectives.

B. Dynamics of an Epidemic

To reflect the dynamics of the disease, models also include incubation periods, length of the infectious period, etc. The size of the group, its geographic dispersal, and the frequency of infective-susceptible contacts all affect the speed with which the epidemic builds to its peak and then dies out. In cities the buildup can take months. This fact has important implications for disease countermeasures.

C. Statistical Behavior of Epidemics

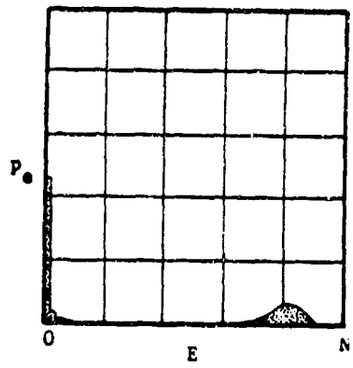
A significant result for our purposes is the Kermack-McKendrick Threshold Theorem[1/] (and its stochastic analog) which states that if the "relative removal rate" of infectives exceeds a threshold value, then the epidemic will die out with only a few cases. If the relative removal rate is too low, then the disease will sweep through nearly the whole population of susceptibles. The exact fraction of susceptibles infected depends on the particular conditions cited above. An example of this behavior is shown in Figure F-1.

E. Effect of Immunization

The second feature of interest is the effect of immunization. If a sufficiently high proportion of the exposed population is immune, an epidemic simply cannot occur.

1/ Bailey, N. T. J. The Mathematical Theory of Epidemics. New York: Hafner Publishing Company, 1957.

The percent of the population which must be immune depends on the type of mixing, the infectiousness of the disease, the size of the group, etc.; but in general it can be less than 100 percent. A striking example in the United States is polio.

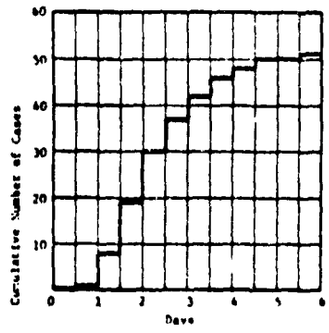


Source: D. G. Kendall. "Mathematical Models of the Spread of Infection," Mathematics and Computer Science in Biology and Medicine, pp. 213-225.

Fig. F-1. The Distribution of the Size, E, of a Stochastic Epidemic; N = Population of Susceptibles

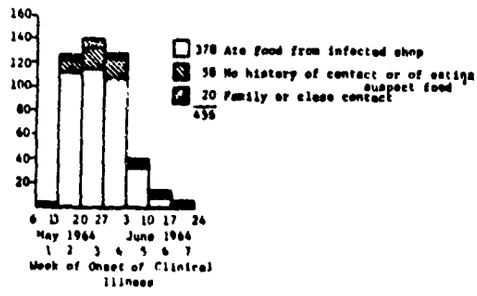
II. FOOD- OR WATER-TO-MAN TRANSMISSION

For food- and water-borne diseases, theory and observation reveal only that the epidemic follows the typical logistic curve (Figure F-2). This shape reflects a distribution of times of ingestion as well as a distribution of incubation periods. The size of the epidemic will depend on the exposure pattern (the whole community's water supply, or only a few contaminated fruits and vegetables), the concentration of the organism (amount of it ingested), and the immunity status. If, in a disrupted environment, boiling or sterilizing drinking water, washing raw fruits and vegetables with mild antiseptics, and cooking food well were strictly observed, enteric infections could be limited. Since this cannot be presumed to be the case initially, we estimate a rather severe threat from gastroenteritis because the organisms are so widespread.



Source: Williams, T. "The Basic Birth-Death Model for Microbial Infections," The Journal of the Royal Statistical Society. Series B, Vol. 27, No. 2, 1965.

Fig. 2a. Distribution of Incubation Periods in Milkborne Outbreaks of Streptococcal Sore Throat in Catskill, New York, U. S. A.



Source: Walker, William. "The Aberdeen Typhoid Outbreak of 1964" Scotland Medical Journal, 1965, pp. 466-79.

Fig. 2b. Frequency of Typhoid Cases by Weeks of Onset and History of Exposure, Aberdeen, Scotland.

Fig. 2

III. VECTOR BORNE DISEASES

The mathematical modeling of disease transmitted to man via a vector host is considerably more complex than for the man-to-man class. It has, however, been successfully carried out for malaria. The field is reviewed by Serfling.[2/] The principal results of interest relate to human and mosquito vector populations in equilibrium (the disease is endemic) and not to the probability that an epidemic occurs when conditions are altered. Thus, current theory was of less value for this class, since as is later shown, mosquito borne diseases are not important.

2/ Serfling, Robert E. "Historical Review of Epidemic Theory," Human Biology, A Record of Research. Vol. 24, No. 3, pp. 145-166.

Appendix G

Estimated Postattack Disease Parameters, Countermeasures, and Preventable Deaths

This appendix contains a description of the procedures used to estimate provisionary disease parameters (Section I), the effect of countermeasures (Section II), and the number of preventable deaths (Section III). These estimates provide the basis for the Disease and Chronic Conditions Submodel.

Appendix G

I. ESTIMATED POSTATTACK DISEASE PARAMETERS

The following definitions and tables are used in deriving estimates of health status:

1. Postattack conditions favoring disease spread conditions after a nuclear attack are very austere and involve disruption of family, food, supplies, sewage treatment, and public health systems in general.
2. Community size (and composition) has an effect on health problems. A community size of 20,000 was assumed. For example, sewage disposal in a very small community may be of little concern up to a point because natural controls of the environment can be sufficient. However, the larger the community the more important becomes human intervention in pollution control. Similarly, the larger the community the more likely is the appearance of one infective. On the other hand, the larger the community the longer it takes the epidemic to peak out, and the lower the percent of population affected if an epidemic occurs.
3. The attack rate for a communicable disease is the percentage infected.
4. Countermeasures considered are as follows:
   a. Medical prophylaxis--the use of antibiotics, antibacterials, vaccine, gamma globulin and antiseptics.
   b. Vector control--the use of chemicals, sanitation, etc. to reduce insect populations, and control of animals which may be the source of infection.
   c. Food, water, sewage and personal hygiene measures--control of infectious agents through the proper storage, distribution, and preparation of food and water. It also includes personal hygiene. Because of the postattack conditions that have been assumed, many of the personal hygiene measures will be very difficult to take. These include avoiding contact with infectives, exposure to the elements, adequate sleep, etc. Quarantine and isolation may be difficult to enforce.
   d. Medical treatment--currently available medical knowledge, procedures, and treatment supplies have been assumed.

5. Expected fatalities = (P_i = Probability of initial infection) x (Probability of epidemic given P_i) x (Expected size of epidemic given that it "takes off") x (Case fatality rate)

Each of the four factors of course varies with the health preparedness measures. For estimated values of the first two factors, see Table G-I.

Table G-I

PROBABILITIES ASSOCIATED WITH DISEASE SPREAD
(POSTATTACK CONDITIONS FAVORING DISEASE SPREAD AND A COMMUNITY SIZE 20,000)

	Probability of at Least One Case Appearing	Probability of an Epidemic Given One Case
Pneumonia	0.95	0.7
Influenza	0.95	0.95
Typhoid	0.05	0.7
Paratyphoid B	0.05	0.7
Dysentery	0.3	0.7
Cholera	0.0005	0.95
Hepatitis	0.7	0.3
Plague	0.3	0.7
Smallpox[1/]	0.005	0.3
Typhus	0.005	0.7
Whooping Cough	0.7	0.7
Measles	0.95	0.3
Diptheria[1/]	0.7	0.7
Gastroenteritis	0.95	0.7
Scarlet Fever[2/]	0.05	0.7
Meningococcal Meningitis	0.05	0.05

[1/] Assuming present rate of vaccination or immunization.

[2/] Includes severe streptococcal sore throat.

Table G-II shows the relative weight of Man-to-Man, Enteric, and Vector Borne modes of transmission for postattack diseases. For example, Typhoid is transmitted in 20% of the cases by Man-to-Man contact and in 80% of the cases by Enteric contact.

Table G-II

THE RELATIVE WEIGHT OF DIFFERENT MODES OF TRANSMISSION OF THE MAJOR POSTATTACK COMMUNICABLE DISEASES
(in percent)

Diseases	Man-to-Man	Enteric	Vector Borne
Pneumonia	100	0	0
Influenza	100	0	0
Typhoid	20	80	0
Parathyphoid B	20	80	0
Dysentery	20	80	0
Cholera	30	70	0
Hepatitis	60	40	0
Plague	50	0	50
Smallpox	100	0	0
Typhus	0	0	100
Whooping Cough	100	0	0
Measles	100	0	0
Diptheria	90	10	0
Gastroenteritis	10	90	0
Scarlet Fever	80	20	0
Meningococcal Meningitis	100	0	0

II. ESTIMATING EFFECTS OF COUNTERMEASURES

The variations in the health system which were examined as countermeasures are:

Medical treatment (biologicals, chemotherapy)--affects the case fatality rate.

Medical prophylaxis (immunization, prophylactic use of antibiotics, antibacterials, antiseptics, and gamma globulin)--affects the expected size of the epidemic and the probability of occurrence of an epidemic. Case fatality rate would also be affected (partially successful immunization mitigates the severity of an attack) but this was not estimated.

Food and water purity, sewage control, and personal hygiene--affects probability of an initial case and expected size of epidemics for enteric infections, and the expected size of an epidemic for man-to-man transmission.

Vector control (reduction of insect populations)--affects probability of an initial case of plague and typhus and the expected size of an epidemic should one occur.

The effects of these health measures on fatalities were estimated singly and combined. Since some diseases have more than one mode of transmission, computing the effect of a countermeasure specific to mode-of-transmission (vector control, say) requires partitioning the disease between two categories. This set of requirements was shown in Table G-III.

One measure of the importance of various communicable diseases postattack is the expected numbers of deaths. Such an estimate is shown in Figure G-1 when there are no countermeasures. Similar figures can be derived for combinations of countermeasures. It is interesting to note that when these values are plotted in a cumulative plot (deaths versus number of causes) the results are consistent with the hypothesis that a small number account for a large share of the disease problems.

On the other hand, prevalence estimates are required to give guidance about medical care requirements and general health status.

The kind and amount of staff and drugs designated to be needed for obtaining minimum case fatality is listed by disease in Table G-IV. The multipliers for this minimum fatality rate in the event that staff or drugs are only half available, or not at all, are given in Table G-V.

Table G-III

ESTIMATED SIZE OF EPIDEMIC AND CASE FATALITY RATES FOR SELECTED COMMUNICABLE DISEASES UNDER POSTATTACK CONDITIONS FAVORING DISEASE SPREAD AND ASSUMING A COMMUNITY SIZE OF 20,000
(% of Population)

Disease	Expected Size of Epidemic (% of Population)															Fatality Rate (% of Cases)					
	A With No Preventive Measures			B With Prophylactic Medical Measures			C With Vector Control			D With Food, Water and Sewage Countermeasures and Personal Hygiene			E With Combined Countermeasures (B,C, and D)			F Untreated			G Treated		
	Low	High	Mean	Low	High	Mean	Low	High	Mean	Low	High	Mean	Low	High	Mean	Low	High	Mean	Low	High	Mean
Pneumonia	1	5	3	1	5	3	1	5	3	1	4	2.5	1	3.5	2.25	10	50	30	5	15	10
Influenza	10	50	30	1	5	3	10	50	30	10	40	25	1	4	2.5	0.5	3	1.75	0.5	3	1.75
Typhoid	10	40	25	0.5	5	2.75	10	40	25	0.5	3	1.75	0.5	2.5	1.5	10	20	15	2	3	2.5
Paratyphoid B	10	40	25	1	10	5.5	10	40	25	1	5	3	1	4	2.5	1	5	3	0.5	1	0.75
Dysentery	4	80	42	0.5	10	5.25	4	80	42	0.5	3	1.75	0.5	2.5	1.5	15	35	25	0.5	1	0.75
Cholera	30	50	40	1	15	8	30	50	40	0.5	5	2.75	0.5	1	0.75	10	75	42.5	5	10	7.5
Hepatitis	1	20	10.5	0.5	10	5.25	1	20	10.5	0.5	3	1.75	0.5	2.5	1.5	0.5	1	0.75	0.5	1	0.75
Plague	10	50	30	1	20	10.5	0.5	5	2.75	10	40	25	0.5	1	0.75	25	75	50	5	10	7.5
Smallpox	1	20	10.5	0.5	1	0.75	1	20	10.5	1	16	8.5	0.5	1	0.75	5	10	7.5	5	10	7.5
Typhus	1	30	15.5	1	5	3	0.5	3	1.75	1	25	13	0.5	1	0.75	10	60	35	3	10	6.5
Whooping Cough	3	6	4.5	0.15	3	1.575	3	6	4.5	3	5	4	0.15	1	0.75	2	3	2.5	0	0.1	0.005
Measles[1]	1	10	5.5	1	10	5.5	1	10	5.5	1	9	5	1	9	5	2	5	3.5	0.5	1	0.75
Diphtheria	10	50	30	0.5	1	0.75	10	50	30	10	40	25	0.5	1	0.75	5	10	7.5	0.5	1	0.75
Gastroenteritis	50	90	70	1	10	5.5	50	40	70	1	5	3	1	4	2.5	5	30	17.5	1	5	3
Scarlet Fever[2]	10	50	30	0	2	1	10	50	30	10	40	25	0	1	0.5	1	3	2	0.5	1	0.75
Meningococcal Meningitis	1	5	3	1	5	3	1	5	3	1	4	2.5	1	3.5	2.25	40	50	45	1	5	3

[1] Assuming vaccine will not be generally available till 1968.

[2] Rates include severe streptococcal sore throats

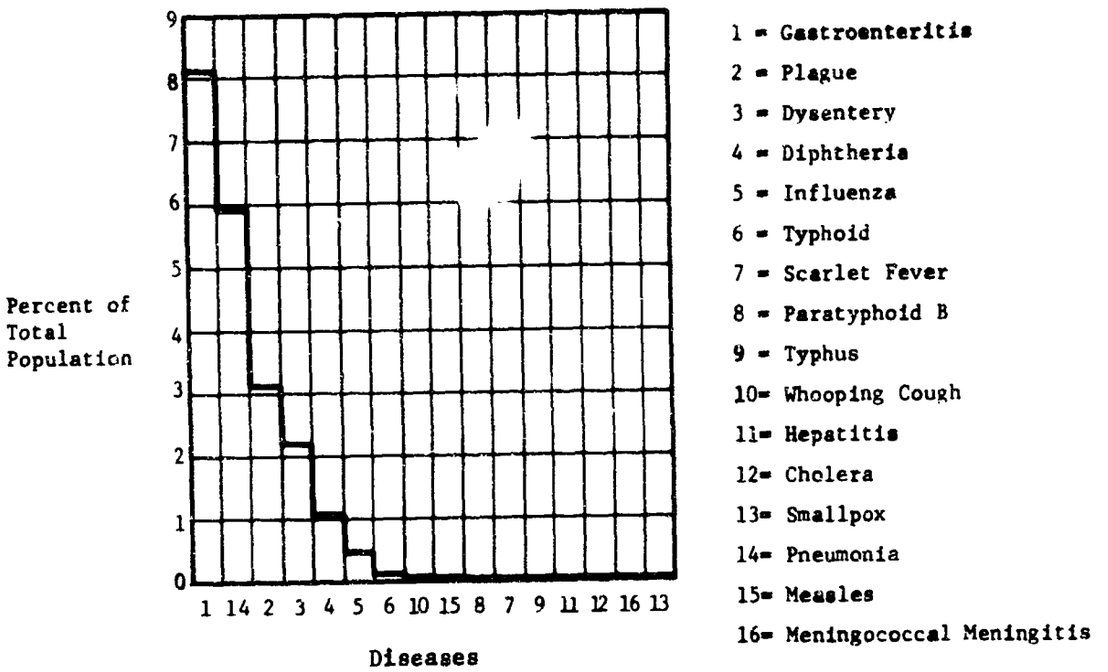


Fig. G-1. Expected Untreated Death Rates by Major Communicable Diseases With No Countermeasures

Table G-IV

GUIDELINE FOR DESIGNATED TREATMENT BY DISEASE

Disease	Kind and Daily Amount of Prof. Staff*			Kind and Daily Amount of Drugs**
Pneumonia	.02P	.10N	.02T	1B
Influenza	.02P	.10N	.02T	1B
Typhoid	.02P	.10N	.02T	(0.5)E
Paratyphoid B	.02P	.10N	.02T	1E
Dysentery	.02P	.10N	.02T	1D
Cholera	.02P	.10N	.02T	1A
Hepatitis	.02P	.10N	.01T	
Plague	.02P	.10N	.02T	2C
Smallpox	.02P	.10N		(0.5)B
Typhus	.02P	.10N	.02T	1C
Whooping Cough	.02P	.10N	.02T	(0.25)B
Measles	.02P	.10N		1B
Diphtheria	.02P	.10N	.02T	1B
Gastroenteritis	.02P	.10N	.02T	1A
Scarlet fever	.02P	.10N	.02T	1B
Meningococcal Meningitis	.02P	.10N	.02T	(0.05)B

* P physician
N nurse
T laboratory technician

} The decimal fraction before the letter indicates the part of a man-day devoted to the care of one patient.

** A Intraveneous infusion--3 pints of Saline
B Penicillin--1,000,000 units
C Tetracyclin--1 gram
D Sulfa--1 gram
E Ampicillin--5 gram

(The number before the letter indicates the number of doses of the drug.)

Table G-V

MULTIPLICATIVE MORTALITY FACTORS FOR DOWNGRADING PROFESSIONAL STAFF OR DRUGS OVER ONE LEVEL *

Disease	1/2 Staff	1/2 Drugs
Influenza	1.7	1.4
Parathyphoid B	1.8	1.5
Dysentery (Shigellosis)	3.0	2.0
Hepatitis (Infectious)	1.5	1.2
Whooping Cough	2.0	1.4
Measles	2.2	2.2
Diptheria	2.5	1.8
Gastroenteritis	3.0	1.8
Scarlet Fever	1.7	1.4

* These factors are to be applied to the individual death rates of a disease when full medical staff is not available or adequate drugs are not available. These multipliers are applied once for 1/2 of the required supplies (or staff) or twice for no supplies (or staff.) For example, Dysentery with 1/2 required staff and no drugs would be 3.0 x 2.0 x 2.0 = 12.0 times the fully treated death rate.

III. ESTIMATION OF PREVENTABLE DEATHS

A more illuminating way to present the results is in terms of "Preventable Deaths." This measure of effectiveness, which is the difference in expected fatalities with and without particular combination countermeasures, shows the necessity of planning emergency medical programs. These results are summarized in Table G-VI.

Table G-VI

EXPECTED PREVENTABLE DEATH RATES AS AFFECTED BY COUNTERMEASURES
(% of Total Population)

Countermeasures		Mode of Transmission			
		Man-to-Man	Food Borne	Vector Borne	Total
Medical Prophylaxis	Without Medical Treatment	3.7	8.5	1.0	13.2
	With Medical Treatment	0.8	1.2	0.2	2.2
Food, Water, Sewage, and Personal Hygiene	Without Medical Treatment	1.9	8.8	0.3	11.0
	With Medical Treatment	0.3	1.3	0.1	1.7
Vector Control	Without Medical Treatment	1.4	0	1.5	2.9
	With Medical Treatment	0.2	0	0.2	.4
Medical Prophylaxis, Food, Water, Sewage, Personal Hygiene and Vector Control	Without Medical Treatment	4.4	9.0	1.6	15.0
	With Medical Treatment	1.0	1.3	0.2	2.5
Medical Treatment	Without Counter-measures	3.9	8.0	1.3	13.2
	With Counter-measures	0.4	0.3	0.1	0.8

Appendix H

Methods of Estimating the Soper-Reed-Frost Parameters from Literature Data for the Disease and Chronic Conditions Submodel

This appendix lists the various methods by which the Soper-Reed-Frost parameters can be estimated for given communities and diseases, using data available in the literature.

Appendix H

Methods of Estimating the Soper-Reed-Frost Parameters From Literature Data

I. INTRODUCTION

There are three ways to assess the contact rate (λ):

1) By using the model represented by equation 3-2 if the values of all other characteristics except one are known from observations.
2) By means of the attack rate if certain conditions are met.
3) By means of the average time interval between successive epidemic waves in the case of recurrent epidemics in large communities if some other characteristics are known.

The within-household contact rates used for this model were determined by a secondary attack ratio (see "2" above).

II. SPECIFIC ASSESSMENTS OF CONTACT RATE

A. Assessment by Means of the Deterministic Chain-Infection Model

Assuming influx of new susceptibles, equation (2) in Chapter III permits the calculation of the contact rate if the values for all other characteristics except one are known from observations. These characteristics are: (s) prevalence of susceptible persons, (i) prevalence of infective persons, (m) birth rate, (a) proportion of the infected persons that becomes infective, and (D) average duration of infectivity in an infective person. If the demographic performance of the model does not conform with reality, alternative models can be used.

B. Assessment by Means of the Crude Attack Rate

This method requires that 100 percent of the community is susceptible at the beginning of an epidemic. This requirement is sometimes met in influenza and adenovirus epidemics. It can be shown that under such circumstances, where at the same time $a=1$, the crude attack rate, when expressed as a proportion of the total population (N), is a measure of the contact rate. Kendall[1/] worked with Soper's

---

1/ Kendall, D. G. "Deterministic and Stochastic Epidemics in Closed Populations," Proceedings. Third Berkeley Symposium on Mathematical Statistics and Probability. Berkeley and Los Angeles, Calif.: University of California, Vol. 4, 1956, pp. 149-165.

continuous-infection model (see chapter III)

$$A = SpI$$

where A is the number of individuals infected during one unit of time. This formula is similar to $\frac{A}{N} = s\lambda i$ since approximately $pN = \lambda$ when N is large enough. The latter equation is the limit of equation (2), chapter III.

$$\frac{A}{N} = s(1-e^{-\lambda i})$$

when λi approaches zero. For most epidemics this is probably a fair approximation.

By means of a mathematical treatment of the Soper model, Kendall arrives at the results given in table H-I.

Table H-I

RELATIONSHIP OF CRUDE ATTACK RATE TO PRODUCT OF CONTACT RATE AND DURATION OF INFECTIVITY IN AN EPIDEMIC STARTING WITH A POPULATION OF 100 PERCENT SUSCEPTIBLES[a/]

Proportion of Population Attacked	Product of Contact Rate and Duration of Infectivity[b/]
.00	1.00
.10	1.05
.20	1.12
.30	1.19
.40	1.28
.50	1.39
.60	1.53
.70	1.72
.80	2.01
.90	2.56
.95	3.15
.98	3.99

a/ Source: Kendall, D. G. "Deterministic and Stochastic Epidemics in Closed Populations," Proceedings, Third Berkeley Symposium on Mathematical Statistics and Probability. Berkeley and Los Angeles, Calif., Vol. 4, 1956, pp. 149-165.

b/ D. G. Kendall used the term "Population as Multiple of 'Threshold' N/ρ," where ρ is equivalent to $1/(Dp)$ in the presently used notation. Since approximately $p = \lambda/N$ (see equation (2) in chapter III), replace the whole column heading by $NDp = ND\lambda/N = D\lambda$.

tion, knowing the proportion of the population attacked and the value for the average duration of infectivity, the value of the contact rate can be calculated.

Because this model does not take into account the influx of new susceptibles by birth and migration, this method is applicable only to acute epidemics of short duration. Under these circumstances, the influx of new susceptibles can be neglected.

C. Assessment by Means of the Time Interval Between Peaks of Recurrent Epidemics

Bartlett[2/] used the Soper model with influx of new susceptibles (Chapter III). In a stochastic stationary process, the values for the number of prevalent susceptibles (S) and prevalent infectives (I) have the expected values $E(S) = S_o$ and $E(I) = I_o$ respectively. By equating the expected values of the first derivatives of S_t and I_t to zero, so that

$$E\left[\frac{(dS)_t}{dt}\right] = 0 \quad \text{and} \quad E\left[\frac{(dI)_t}{dt}\right] = 0 ,$$

the following equations are obtained:

$$D = \frac{I_o}{M} \qquad \text{and} \qquad p = \frac{1}{S_o D} . \tag{H-1}$$

These equations are identical to the deterministic ones of the Soper model where S_o and I_o are equilibrium values.

Moreover, Bartlett[3/] assumed an influx of new infectives into the community from outside at the rate of ε persons per time unit or of the proportion $\frac{\varepsilon}{N}$ of the community. Now let $\frac{\varepsilon}{N}$ [4/] be small so that the delay in the growth of an epidemic wave after the influx of an infective from the outside can be neglected. (The delay in growth should not be confused with the delay or waiting time since the end of the previous epidemic before an infective immigrates from the outside; the latter waiting time gets longer as $\frac{\varepsilon}{N}$ becomes smaller.) Bartlett's treatment of the equations relevant to this situation implies that the probability density of the distribution of the delay time before the start of a new epidemic wave is

$$r(T-1)T^{r-1}e^{-r(T-1)}$$

2/ Bartlett, M. S. "Measles Periodicity and Community Size," Journal of Royal Statistical Society. Series A, Vol. 120, 1957, pp. 48-60.

3/ Bartlett, M. S. Stochastic Processes. Cambridge University Press, 1955.

4/ Bartlett (1955) actually wrote ε instead of ε/N, but since in his reasoning the characteristic seems to be independent of the community size N it is better represented by ε/N if ε is proportionate to N. He made the latter assumption in 1957.

where

$$T = m \lambda D t \,, \qquad \text{(H-2)}$$

$$r = \frac{\left(\frac{\epsilon}{N}\right) N}{m \lambda D} \qquad \text{(H-3)}$$

t = duration of time since the end of the previous epidemic wave, when at that time therewere no susceptibles

This distribution has a mode at

$$T = 1 + \frac{1}{r} \,. \qquad \text{(H-4)}$$

Thus the time $\tilde{t}$ corresponding to this mode can be obtained by substituting equations (H-2) and (H-3) into (H-4):

$$m \lambda D \tilde{t} = 1 + \sqrt{\frac{m \lambda D}{\frac{\epsilon}{N} N}}$$

$$\tilde{t} = \frac{1}{m \lambda D} + \sqrt{\frac{1}{\left(\frac{\epsilon}{N}\right) N m \lambda D}} \qquad \text{(H-5)}$$

If N is large enough, equation (H-5) can be replaced by

$$\tilde{t} = \frac{1}{m \lambda d} \,. \qquad \text{(H-6)}$$

Equation (H-1) can be multiplied on both sides by N

$$p N = \frac{1}{\frac{S_o}{N} \cdot D} \quad \text{so that } \lambda = pN = \frac{1}{s_o D}$$

The results can be substituted into equation (H-6) so that

$$\tilde{t} = \frac{s_o}{m} \qquad \text{(H-7)}$$

where $\tilde{t}$ is the predicted mode of the duration of the time interval between two consecutive epidemic waves.

Two questions need to be answered before this formula can be applied:

a) <u>What is the minimum population size N compatible with the use of the abbreviated formulas (H-6) and (H-7)</u>?

Bartlett[5/] reported the buildup of an artificial epidemic process extended in space as well as in time. He found that this simulation predicted that

5/ Bartlett, M. S. "Measles Periodicity and Community Size," Journal of Royal Statistical Society. Series A, Vol. 120, 1957, pp. 48-60.

relatively isolated towns similar to certain British towns need more than 200,000 inhabitants for measles to remain endemic without an influx of infective patients. When he tested the prediction with observations of towns, he found that they did not need an influx from outside if the number of inhabitants was more than 250,000 or 300,000. Thus, the fit between prediction and observation was fairly good and this supports the realism of Bartlett's model.

Data used by Bartlett confer the impression that a relatively isolated town needs more than 100,000 inhabitants for its average interval between consecutive epidemic peaks to be independent of population size. Hence, for such a population size the second term of equation (H-5) is of minor importance relative to the first term.

b) Are equations (H-6) and (H-7) applicable when $\alpha < 1$?

For different values of α in table H-11, the prevalence of infectives is given for consecutive time units following the artificial event where 100 percent of the community members are simultaneously infected. Calculation is based on the assumption that the intensity of infectivity is equally distributed over the total duration of infectivity. When the beginning of the infective period coincides with the highest intensity of infectivity, the influence of the size of α on the prevalence of infectives is less marked; in general, influenza is such a case (Woodall, et. al.[6/]). The influence of the size of α is still less if the infections, rather than taking place simultaneously, are distributed more evenly over time. Finally, in the equilibrium state in which the number of new infections is equal for all points in time, there is complete independence between the size of α and the prevalence of infectives as long as the product αD remains constant. In many cases where a stationary process is reached and the amplitude of the oscillations of s and i around the expected values is small. Equations (H-6) and (H-7) also seem applicable to diseases (like tuberculosis) with a value for α that is appreciably smaller than one. In other diseases (like diphtheria), if the value of α is not too small, a much larger amplitude of the oscillations seems to be compatible with use of equations (H-6) and (H-7).

D. Assessment of the Within-Household Contact Rate by Means of the Secondary Attack Rate

In considering a population 100 percent susceptible to a disease, the secondary attack ratio (B), expressed as a proportion of the population, is here:

$$B = \frac{\text{Number of persons secondarily attacked}}{\text{Number of persons exposed}}$$

$$= \frac{\text{Number of persons secondarily attacked in the household}}{\text{Number of persons (all susceptible) present in the household}}$$

6/ Woodall, J., K. C. K. Rowson, and J. C. McDonald. "Age and Asian Influenza," British Medical Journal, No. 5108, Vol. 2, 1958, pp. 1316-1318.

Table H-II

TIME SEQUENCE OF THE PREVALENCE OF INFECTIVES AFTER SIMULTANEOUS INFECTION OF ALL MEMBERS OF A COMMUNITY, FOR DIFFERENT PROPORTIONS OF THE INFECTED PERSONS THAT BECOME INFECTIVE, THE OVERALL NONSPECIFIC SUSCEPTIBILITY BEING EQUAL IN ALL INSTANCES

Proportion of infected persons that becomes infective	Duration of infectivity (in time units)	Prevalence of infective persons* (assuming 100 percent simultaneously that the intensity of infectivity is distributed equally over the whole duration of infectivity)						
		Duration of time periods (in time units)						
(a)	(D)	0.0-0.9	1.0-1.9	2.0-2.9	3.0-3.9	4.0-4.9	5.0-5.9	6.0-6.9
1.0	1.000	1.00	0.00	0.00	0.00	0.00	0.00	0.00
0.9	1.111	0.90	0.10	0.00	0.00	0.00	0.00	0.00
0.8	1.125	0.80	0.20	0.00	0.00	0.00	0.00	0.00
0.7	1.429	0.70	0.30	0.00	0.00	0.00	0.00	0.00
0.6	1.667	0.60	0.40	0.00	0.00	0.00	0.00	0.00
0.5	2.000	0.50	0.50	0.00	0.00	0.00	0.00	0.00
0.4	2.500	0.40	0.40	0.20	0.00	0.00	0.00	0.00
0.3	3.333	0.33	0.33	0.33	0.00	0.00	0.00	0.00
0.2	5.000	0.20	0.20	0.20	0.20	0.20	0.20	0.00
0.1	10.000	0.10	0.10	0.10	0.10	0.10	0.10	0.10

$$= \frac{(A)_{0,D}}{n-1} \qquad \text{(H-8)}$$

where $(A)_{0,D}$ is distinguished from $A = \frac{i}{\alpha D}$ (Chapter III) by being observed over the time interval 0,D instead of the usual unit of time (D is the duration of infectivity in an infective person).

If only households with one primary case are considered,

$$B = (p')_{0,D} \qquad \text{(H-9)}$$

where $(p')_{0,D}$ is similarly distinguished from p' by using the probability of at least one contact between any two specified persons in the household during the time interval 0,D.

Apply equation (3), Chapter III, to one of the algebraic definitions of e, the base of the natural logarithm:

$$\left[1 - (p')_{0,D}\right]^{n-1} = e^{-D\lambda'} \qquad \text{(H-10)}$$

Substituting equation (H-9) into (H-10),

$$(1 - B)^{n-1} = e^{-D\lambda'} \quad \text{or} \quad \lambda' = -\frac{n-1}{D} \ln(1-B). \qquad \text{(H-11)}$$

Thus, if n, D, and B are known, the within-household contact rate can be calculated.

In summary, the conditions to be met are: (1) The household should contain only susceptibles except for the primary case; (2) The secondary cases should have been infected by the primary case only, not by each other nor by outsiders; (3) All subclinical infections should be reported.

III. ASSESSMENT OF THE NONSPECIFIC HOST RESISTANCE

If it can be assumed that there are no deaths among the susceptibles and that the population is stationary and stable (constant birth rate equals the constant death rate),

$$\frac{i}{\alpha D} = m\left(1 - e^{\frac{-\lambda i}{m}}\right) \quad \text{so that} \quad \alpha D = \frac{i}{m\left(1 - e^{\frac{-\lambda i}{m}}\right)} .$$

Appendix I

Determinants of the Contact Rate in the Disease and Chronic Conditions Submodel

This appendix describes and summarizes nine environmental attributes and their interrelationships which are *a priori* likely to influence the rate of epidemiologic contact between individuals in the Soper-Reed-Frost model.

Appendix I

Determinants of the Contact Rate in the Disease and Chronic Conditions Submodel

Several behavioral and environmental attributes are likely to influence the level of the contact rate. Nine of these attributes will be considered in this Appendix; i.e., radiation, crowding within the household, size of the household, relative humidity, temperature level, temperature changes, season, ventilation and air pollution. In order to assess the relative effects of these nine attributes, it is necessary to measure the contact between individuals in the Soper-Reed-Frost model. If one assumes that persons at risk are known to be susceptible, this contact rate might be measured within the household as a secondary attack ratio and in the community as a crude attack rate. The restriction of classifying the persons at risk as susceptible or nonsusceptible usually demands skin tests (for tuberculosis and diptheria), serological tests (for influenza) or reliable histories (for measles) in diseases conferring lifelong immunity. The following virus diseases, however, have the advantage of not needing these tests to provide data for computing the contact rate: common cold, adenovirus type 4, and influenza type A.

The common cold can be caused by a number of viruses; each virus probably differs in its invasiveness and communicability. Hence, in order to apply observations on the quantitative relationship between crowding and contact rate of the common cold to the quantitative relationship of other respiratory diseases, it is assumed that the numerous common cold viruses are fortuitously randomly distributed among all population groups as observed or predicted. Under this assumption the common cold can be viewed as one disease with short-term immunity to which large population sections are susceptible.

Adenovirus type 4, a disease producing lifelong immunity, rarely attacks non-military populations; thus it can be assumed without much investigation, that certain young age groups are susceptible (nonimmune) to this disease.

Studies of influenza type A (by Davenport, Francis, Hennessy, Hilleman, and Jensen)[1-8] show that, throughout the world, persons of the same age have largely identical immunological experience with the various "families" and strains of this virus type. Thus, like for adenovirus type 4, it is sometimes justified to make sweeping generalizations concerning the susceptibility of an entire population when a new "family" or strain emerges by antigenic mutation.

A brief discussion of the nine attributes that influence the level of contact rate is presented below.

1. Radiation

If radiation influences the contact rate, it is in the direction of an increase. However, in view of present knowledge, it is unlikely that this influence will be large compared to the change in susceptibility.

2. Crowding Within Household (common cold)

Brimblecombe et al.[8] found that differences in degree of crowding are correlated with differences in the secondary attack ratio (this ratio can be an index of within-household contact rate under given circumstances, Appendix H.

1/ Davenport, F. M., A. V. Hennessy, and T. Francis. "Epidemiologic and Immunologic Significance of Age Distribution of Antibody to Antigenic Variants of Influenza Virus," Journal of Experimental Medicine, Vol. 98. 1953, pp. 641-656.

2/ Francis, T. "A Serological Recapitulation of Human Infection with Different Strains of Influenza Virus," Transactions of the Association of American Physicians, Vol. 66, 1953, pp. 231-239.

3/ Jensen, K. E., and T. Francis. "The Antigenic Composition of Influenza Virus Measured by Antibody Absorption," Journal of Experimental Medicine, Vol. 98, 1953, pp. 619-639.

4/ Hilleman, M. R., and J. H. Werner. "Recovery of New Agent from Patients with Acute Respiratory Illness," Proceedings of the Society of Experimental Biology and Medicine, Vol. 85, 1954, pp 183-188.

5/ Hennessy, A. V., F. M. Davenport, and T. Francis. "Studies of Antibodies to Strains of Influenza Virus in Persons of Different Ages in Sera Collected in a Postepidemic Period," Journal of Immunology, Vol. 75, 1955, pp. 401-409.

6/ Davenport, F. M., and A. V. Hennessy. "A Serologic Recapitulation of Past Experiences with Influenza A; Antibody Response to Monovalent Vaccin," Journal of Experimental Medicine, Vol. 104, 1956, pp. 85-97.

7/ Jensen, K. E. "The Nature of Serological Relationships Among Influenza Viruses," Advances in Virus Research, Vol. 4, 1957, pp. 279-310.

8/ Brimblecombe, F. S. W., et al. "Family Studies of Respiratory Infections," British Medical Journal, No. 5063, Vol. 1, 1958, pp. 119-128.

In their study, if one or two rooms were available for a family of five, the secondary attack rate averaged 17.4 percent; if four or more rooms were available for such a family, the rate averaged 13.5 percent. If this association between crowding and secondary attack rate would remain the same when other factors showing association with the secondary attack rate are taken into account, such an association under the same assumptions could be applied to other respiratory diseases.

3. Size of the Household (common cold)

An English study on the distribution of secondary infections of the common cold over the households of a rural community gives some clues to the influence of size and composition of the household on this distribution.

Lidwell and Sommerville[9/] observed distributions of secondary infections in the village of Bowerchalke near Salisbury, England and classified the households according to size and combination of age groups ("adules", "school children", "infants") belonging to the household. They performed two operations for each class of households separately: (1) They plotted a frequency histogram of the number of secondary infections observed, and (2) they determined the single value of the within-household contact rate p' best fitting to all observations in this class. Using this p' value and a random variable to simulate within-household infection, they plotted a frequency histogram of the number of secondary infections thus predicted by the Reed-Frost model. In superimposing the observed and predicted histograms thus obtained, Lidwell and Sommerville found a good fit for each class of households.

The concept of contact rate applies not only to household classes but also to individuals within the household. For example, if a household is made up of four individuals with p' values of 0.1, 0.2, 0.3, and 0.4, the members share a "household p' " equal to the arithmetic average of the figures (0.25).

The members of a number of households can be stratified according to "family status" (father, mother, older or younger school child, toddler). An index of the average individual p' values of such strata is the "relative communicability ratio" devised by Brimblecombe et al. Ratios for the five mentioned categories of family status were assessed in a study of London families attending Paddington Green Children's Hospital Clinic.

9/ Lidewell, O. M. and T. Sommerville, "Observations on the Incidence and Distribution of the Common Cold in a Rural Community During 1948 and 1949," Journal of Hygiene, Vol. 49, 1951, pp. 365-381.

Table I-I

BEST-FITTING VALUES OF THE RATE OF CONTACT "WITHIN HOUSEHOLDS" (p') FOR COMMON COLD BY TYPE OF HOUSEHOLD
Based on Observation in English Families, 1950-1958[a]

Type of Household					
Number of Members	Age Group of Members	Best-fitting-values[b] $(p')_{0,D}$	Values[c] (p') Corresponding to best-fitting $(p')_{0,D}$	(p') Values Adjusted for[d] "family status" p'*	p'*(n-1) = λ
2	Adult only	.205	.115	.111	.111
3	Adult only	.273	.160	.155	.310
3	Adult School-age	.201	.111	.092	.183
3	Adult Infant	.284	.167	.151	.302
4	Adult only	.146	.079	.076	.229
4	Adult School-age	.246	.141	.108	.324
4	Adult Infant	.263	.156	.137	.411
4	Adult School-age Infant	.166	.090	.074	.221
6	Adult School-age	.164	.090	.069	.345
7	Adult School-age	.161	.088	.069	.414
7	Adult School-age Infant	.157	.078	.061	.368

[a] Brimblecombe et al.[10]; Lidwell and Sommerville[11].

[b] The best-fitting $(p')_{0,D}$ values (within household contact rate as measured over a time duration D) were obtained by Lidwell and Sommerville.

[c] It has been assumed that the duration of infectivity D is two time units (incubation periods).

[d] The adjustment was performed by dividing the within-household contact rate p' by the arithmetic average of the "relative communicability ratios" (Brimblecombe et al.) attached to the "family status" as occupied by the household numbers concerned. The age group classification of Lidwell and Sommerville does not completely coincide with the "family status" stratification of Brimblecombe et al. The two sets of data were made comparable by guessing the most likely family status occupied by the age groups examined by Lidwell and Sommerville.

[10] "Family Studies of Respiratory Infections," op. cit.

[11] "Observations in the Incidence and Distribution ...", op. cit.

If this index is applied to the best-fitting p' values obtained by Lidwell and Sommerville, the result is a "p' adjusted for family status". These adjusted p' values can be transformed into contact rates (compare equation (3), Chapter III); thus, the existence or absence of an association between contact rate and household size can be ascertained.

If p' is small enough, the within-household contact rate λ' is equal to $p'(n-1)$. Lidwell and Sommerville's best-fitting p' values adjusted for family status can be inserted into this equation, and the estimated line of regression of the resulting λ' values on the household size n can be assessed. This regression is significant at the 5 percent level according to the customary F-test; the slope of the estimated regression line is +0.041. A point estimate of the coefficient of variation of the contact rate is 0.329. When the household class of two adults only (who presumably spend much of their time outside of their household) is omitted, the regression is not significant and the coefficients of variation of the contact rate is 0.255 (see table I-I). This finding supports the contention that the contact rate is approximately independent of community size.

4. Relative Humidity

In a survey of the common cold, Buckland and Tyrell[12/] showed in a medium suspended in the air that rhinoviruses survive better in higher humidities. On the other hand, Hope-Simpson[13/] recorded an incident during a poliomyelitis epidemic in Denmark in which the breathing apparatus used for paralyzed cases produced dry respiratory air resulting in respiratory infections. These infections ceased immediately after correction of the respiratory air humidity. Hope-Simpson[14/] also mentioned that in a humid (humidity constantly 80 percent) cheese factory in Wales the morbidity rate due to common cold was less than 50 percent of the rate in a control group.

If such findings could be confirmed under different conditions of observation, it would seem to indicate that, in the case of relative air humidity, the response of the host has a larger effect on contact rate than the survival chances of the organism when suspended in the air.

12/ Buckland, F. E. and D. A. J. Tyrrell. "Loss of Infectivity on Drying Various Viruses," Nature, Vol. 195, 1962, pp. 1063-1064.

13/ Simpson, R. E. Hope. "Common Respiratory Diseases--Symposium," Royal Society of Health Journal, Vol. 78, 1958, pp. 593-599.

14/ Simpson, R. E. Hope. "Discussion on the Common Cold," Proceedings of the Royal Society of Medicine, Vol. 51, 1958, pp. 267-271.

Buckland and Tyrell[15] showed that unspecified adenovirus suspended in air survives better under high relative humidity. However, as pointed out for the common cold, the quantitative effect on contact rate by virus survival in air suspension, when the relative humidity is varied, is probably less than the effect of host susceptibility.

Hemmes et al.[16] and Harper[17] observed that aerosols of cultures of influenza type A remain viable longer if the relative humidity is lower: after one hour at room temperature the survival rate was 10 times higher at a relative humidity of 35 percent than at 65 percent. Buckland and Tyrell[15] confirmed this observation. As pointed out for the common cold and adenovirus 4, the quantitative importance of the virus survival in air suspension is probably smaller than the importance of host susceptibility, in terms of the effect of either one of these characteristics on the contact rate under various levels of relative humidity. For influenza A, however, both effects have the same direction. Choudhury[18] compared an urban, exposed and a rural, closed community during the 1957 summer influenza epidemic in India. He concluded that a relative humidity of 40-70 percent seemed to favor the febrile influenza catarrhs and that 80 percent or more seemed to disfavor them. This observation agrees with those of Buckland and Tyrell and of Hemmes.

5. Environmental Temperature Level

Andrewes[19] experimentally chilled volunteers and infected them with virus cultures obtained from common cold patients; he found that the attack rate did not differ from that of non-chilled control volunteers. Dowling et al.[20] conducted similar experiments with similar results. However, in one instance

---

15/ "Loss of Infectivity on Drying Various Viruses," op. cit.

16/ Hemmes, J. H., K. C. Winkler, and S. M. Kool. "Virus Survival as a Seasonal Factor Influenza and Poliomyelitis," Nature, Vol. 188, 1960, pp. 430-431.

17/ Harper, G. J. "Airborne Micro-Organisms' Survival Test with Four Viruses," Journal of Hygiene, Vol. 59, 1961, pp. 479-486.

18/ Choudhury, A. K. "Some Epidemiological Considerations of Influenza 1957," Calcutta Medical Journal, Vol. 59, 1961, pp. 231-236. As quoted in: American Institute of Biological Sciences: An Annotated Bibliography of Influenza with Indexes. Published quarterly. Washington, D. C.: 1957-1963.

19/ Andrews, C. H. "Adventures Among Viruses. III. The Puzzle of the Common Cold," New England Journal of Medicine, Vol. 242, 1950, pp. 235-240.

20/ Dowling, H. W., G. G. Jackson, and T. Inouye. "Transmission of the Experimental Common Cold in Volunteers. II. The Effect of Certain Host Factors Upon Susceptibility," Journal of Laboratory and Clinical Medicine, Vol. 50, 1957, pp. 516-525.

they did find an association: female volunteers in the middle third of their menstrual cycle had higher attack rates when chilled. Roden,[21] in experiments similar to those of Andrewes and Dowling, found that the attack rates were slightly higher in the summer than in the winter and that this difference was more marked among male than female volunteers.

Hemmes[22] and Harper[23] showed that for Influenza A the association between temperature level and virus survival in aerosols is such that variations within the range of prevailing indoor temperatures entail only small changes in survival rates.

6. Environmental Temperature Change (common cold)

Van Loghem[24] found that the attack rate of common colds is increased when there is a drop in the outdoor temperature level. Since the indoor temperature level certainly was more constant than the outdoor and since low humidity of the indoor air was not a problem (central heating systems were not yet in use), the change in temperature as a person went outdoors might well have been the responsible factor. Furthermore, current knowledge (Tromp)[25] concerning the physiological allergy reactions of the respiratory tract suggests that a sudden decrease in temperature promotes the spreading of organisms by the infective host through sneezing.

7. Season

Hilleman et al.,[26,27] found that the primary attack rate of adenovirus type 4 among susceptible Army recruits differed by season. The contact rate derived

---

21/ Roden, A. T. "Variations in the Clinical Pattern of Experimentally Induced Colds," Journal of Hygiene, Vol. 61, 1963, pp. 231-246.

22/ Hemmes, J. H. Thesis. Utrecht (1959), as quoted by Hemmes et al. in: "Virus Survival as a Seasonal Factor in Influenza and Poliomyelitis," op. cit.

23/ "Airborne Micro-organisms' Survival Test With Four Viruses," op. cit.

24/ Van Loghem, J. J. "An Epidemiological Contribution to the Knowledge of the Respiratory Diseases," Journal of Hygiene, Vol. 28, 1928, pp. 33-54.

25/ Tromp, S. W. "Biometeorological Aspects of Respiratory Diseases." Paper read at Air Pollution Medical Research Conference, Los Angeles, American Medical Association, March 2-4, 1966.

26/ Hilleman, M. R., et al. "Epidemiology of RI (RI-67) Group Respiratory Virus Infections in Recruit Populations," American Journal of Hygiene, Vol. 62, 1955, pp. 29-42.

27/ Hilleman, M. R., et al. "Epidemiologic Investigations with Respiratory Disease Virus RI-67," American Journal of Public Health, Vol. 45, 1955, pp. 203-210.

from the primary attack rate (assuming that 80 percent of the new recruits were susceptible) was about twice as large in winter as in summer. This difference could be due to crowding, ventilation, temperature change, relative humidity of indoor air, concomitant pathology like common cold or allergy (sneezing), or a combination of some or all of these factors.

The geographical description of the influenza A epidemic of 1957-1958 suggests that the contact rates were higher in winter and early spring than in summer and early fall (see, for instance, Langmuir[28/]). Again, this association can be interpreted in terms of ventilation, temperature change, relative humidity of indoor air, concomitant pathology like common cold or allergy (sneezing), or a combination of some or all of these factors.

8. Ventilation (tuberculosis)

To determine the infectivity of the air in tuberculosis hospital wards, Riley[29/] titrated this air, using susceptible guinea pigs as an indicator. He found agreement between the amount of air needed to infect a guinea pig and the amount of air inhaled by Scandinavian student nurses before their tuberculin-negative reaction converted. This finding seems to support the notion that increased ventilation of the human environment decreases the contact rate for tuberculosis.

9. Air Pollution (common cold)

McCarroll et al.[30,31/] in a family study in New York City demonstrated a positive correlation between sulfoxide content of the air and crude attack rate of acute respiratory disease (mainly common cold). These data are inconclusive as to whether the contact rate or the host susceptibility is primarily affected.

28/ Langmuir, A D. "Epidemiology of Air-Borne Infection," Bacteriological Reviews, Vol. 25, 1961, pp. 173-181.

29/ Riley, R. L. "Air-Borne Pulmonary Tuberculosis," Bacteriological Reviews, Vol. 25, 1961, pp. 243-248.

30/ McCarroll, J. R., E. J. Cassell, W. I. Ingram, and D. W. Wolter, "Health and the Urban Environment: Health Profiles Versus Environmental Pollutants," American Journal of Public Health, Vol. 56, 1966, pp. 266-275.

31/ McCarroll, J. R., E. J. Cassell, D. W. Wolter, J. D. Mountain, J. R. Diamond, and I. M. Mountain. "Air Pollution and Illness in a Normal Urban Population," Archives of Environmental Health, Vol. 14, 1967, pp. 178-184.

Appendix J

Determinants of Host Susceptibility in the Disease and Chronic Conditions Submodel

This appendix describes five host attributes which *a priori* are likely to influence the host susceptibility which determines in turn the proportion of infected individuals who become infective themselves (the "Infective Ratio.")

Appendix J

Determinants of Host Susceptibility in the Diseases and Chronic Conditions Submodel

The bacterial diseases--tuberculosis, diptheria, and whooping cough--and various virus diseases such as measles can provide data especially useful for the estimation of values for the host susceptibility.

In the case of tuberculosis, indices for the prevalence of infectives (bacterial cultures, direct smears, chest X-rays) are readily available for many communities. There is, however, a caution to be observed in analyzing tuberculosis--especially in rural areas with a mild or warm climate--due to the prevalence of aspecific acid-fast infection which confers a degree of cross-immunity. (Amplification: If the small tuberculin reactions in the Medical Research Council study[1/] are caused by aspecific acid-fast organism, these organisms would reduce the risk of becoming a tuberculous case to one-third of its original value.)

In comparing these various countries at different points in time, allowance should also be made for differences in sensitivity of the index used. (Amplification: Data are available from Africa (Wiles and Rabie[2/]; Dubovsky[3/]; World Health Organization[4/]) from New Guinea (Wijsmuller[5,6/]; Hanegraaf[7/]) and from the United States

---

1/ "B. C. G. and Vole Vacillus Vaccines in the Prevention of Tuberculosis in Adolescents. First report to the Council," British Medical Journal, No. 4964, Vol. 1, 1956, pp. 413-427.

2/ Wiles, F. J. and C. J. Rabie. "Tuberculin and X-Ray Surveys in the Transkey," South African Medical Journal, Vol. 29, 1955, pp. 866-868.

3/ Dubovsky, H. "Mass Miniature X-Ray and Tuberculin Survey in Orange Free State and North Cape Colony," South African Medical Journal, Vol. 29, 1955, pp. 992-997.

4/ Tuberculosis Survey in the Somalilands (1956), Nigeria (1957), Basutoland, Bechuanaland and Swaziland (1958), Ibadan, Nigeria (1958), Ghana (1958), Uganda (1959), Sierra Leone (1959). Copenhagen: World Health Organization Tuberculosis Research Office (year of publication as indicated above).

5/ Wijsmuller, G. Tuberculosis Survey Reports to the Government of Netherlands, New Guinea. Unpublished. Hollandia: 1956-1961.

6/ Wijsmuller, G. Naturally Acquired Tuberculin Sensitivity in New Guinea. Thesis. Amsterdam: 't Koggeschip, 1963.

7/ Hanegraaf, J. A. C. Report to the United Nations' Temporary Executive Authority in West New Guinea Concerning Tuberculosis Survey, Japan. Unpublished. Hollandia: 1962.

and Europe in the preantibiotic era (Burke et al.[8] ; Cochrane et al.[9] ; Borgen et al.[10] ; Refsum[11] ; Sigurdsson and Edwards[12]; Comstock and Sartwell[13]). These data point to levels of the nonspecific host susceptibility to tuberculosis which are roughly 10 times higher in the underdeveloped areas compared with United States and Europe.)

For diphtheria, indices for "equilibrium values" of the prevalence of infectives are difficult to obtain because they demand periodic naso-pharyngeal cultures of populations over a long time. (Amplification: Murray[14] gives prevalence figures for Bantus, South Africa. Gill[15] ; Grossman[16] ; Schuman and Doull[17]; Strebbins[18] ; and

---

8/ Burke, M. H., H. C. Schenk, and J. A. Thrash. "Tuberculosis Studies in Muscogee County, Georgia. II. X-Ray Findings in a Community-Wide Survey and Its Coverage as Determined by a Population Census," Public Health Reports, Vol. 64, 1949, pp. 263-290.

9/ Cochrane, A. L., J. G. Cox, and T. F. Jarman. "Pulmonary Tuberculosis in the Rhondda Fach. An Interim Report of a Survey of a Mining Community," British Medical Journal, No. 8789, Vol. 2, 1952, pp. 843-853.

10/ Borgen, L., S. N. Meyer, and E. Refsum. "Mass Photogluorography, Tuberculin Testing and B. C. G. Vaccination in the District of Akar, Norway, 1947-1949," Acta Tuberculosis Scandinavica, Vol. 25, 1950, pp. 327-355.

11/ Refsum, E. "Mass Investigation by Photofluoroscopy," Acta Tuberculosis Scandinavica, Vol. 27, 1952, pp. 288-302.

12/ Sigurdsson, S. and P. Q. Edwards. "Tuberculosis Morbidity and Mortality in Iceland," World Health Organization Bulletin, Vol. 7, 1952, pp. 153-169.

13/ Comstock, G. W. and P. E. Sartwell. "Tuberculosis Studies in Muscogee County, Georgia. IV. Evaluation of a Community-Wide X-Ray Survey on the Basis of Six Years of Observation," American Journal of Hygiene, Vol. 61,1955, pp. 261-285.

14/ Murray, J. F. "Diphtheria Amongst the Bantu," Journal of Hygiene, Vol. 43, 1943, pp. 159-169.

15/ Gill, D. G. "Schick Tests and Carrier Surveys in White School Children, Alabama, 1937-1938," American Journal of Public Health, Vol. 30, Supplement, 1940, pp. 25-27.

16/ Grossmann, W. "A Schick Test and Diphtheria Carrier Survey of White Children in Virginia (Richmond)," American Journal of Public Health, Vol. 30, Supplement, 1940, pp. 8-15.

17/ Schuman, L. M. and J. A. Doull. "Diphtheria Infection and Morbidity in Cleveland, 1937-1939," American Journal of Public Health, Vol. 30, Supplement, 1940, pp. 16-24.

18/ Strebbins, E. L. "Diphtheria Immunity and Carrier Surveys in New York State," American Journal of Hygiene, Vol. 30, Supplement, 1940, pp. 36-41.

Frost et al.[19] give data for the United States which are reasonably free from previous specific vaccination. One gains the impression that the level of the non-specific host susceptibility is roughly nine times higher in Bantus than in the United States. This conclusion has the important proviso that the selective factors operating in Murray's work are not known.)

For whooping cough mortality reporting seems the only practical index of morbidity or infectivity. Since the reporting of whooping cough mortality is very inadequate for regions not providing specific vaccination, a comparison between the present prevalence in these regions and Europe or United States before the vaccination era is impossible.

In most viral diseases, the proportion a of infected persons that become infective is one, and the average duration D of infectivity is farily constant.

1. Radiation Effects

References on radiation are given in literature surveys by W. H. Taliaferro et al.[20] and by R. D. Stoner et al.[21,22] for the U. S. Armed Forces Epidemiological Board. For viral, rickettsial, bacterial, and protozoan infections as well as helminthic infestations after radiation of the host, the following phenomena (insofar as these have been assessed from the literature) contribute to increased host susceptibility:

a) Decreased antibody response.

b) Increased hypersensitivity to antibiotics.

c) Increased susceptibility to toxins.

d) Decreased effectiveness of cellular defense mechanisms.

e) Decreased effectiveness and sometimes increased harm from immunizing agents.

19/ Frost, W. H., et al. "Diphtheria in Baltimore, a Comparative Study of Morbidity, Carrier Prevalence and Antitoxic Immunity in 1921-1924 and 1933-1936," American Journal of Hygiene, Vol. 24, 1936, pp. 568-585.

20/ Taliaferro, W. H., L. G. Taliaferro, and B. N. Jaroslow. Radiation and Immune Mechanisms. New York: Academic Press, 1964.

21/ Stoner, R. D., M. W. Hess, and V. P. Bond. Radiation and Infection: An Annotated Bibliography. Washington, D. C.: Armed Forces Epidemiological Board, Commission on Radiation and Infection, 1965.

22/ Stoner, R. D. Radiation and Infection: An Annotated Bibliography, Supplement I. Upton, N. Y.: Medical Research Center, Brookhaven National Laboratory, 1967.

In view of parallel experiences where protein malnutrition, instead of radiation, increases the host susceptibility (see appendix K-TAB), it is likely that the five just mentioned phenomena operate synergistically. Also, when there is multiple infection (which, in the case of commensals, is the rule rather than the exception), the various organisms are likely to act synergistically to increase the host susceptibility even more.

2. Nutritional Status

According to Scrimshaw, Taylor and Gordon's survey[23] of 378 articles and books on the subject "interaction of nutrition and infection", there seems to be a marked association between nutritional status and nonspecific host resistance against bacterial disease, but little or no association in the case of viral disease.

There is a difference in concept between the chronic protein malnutrition observed in underdeveloped countries and the acute caloric malnutrition observed during periods of war. In the former case in which the caloric intake is usually more adequate than the protein intake, there is a clear association between susceptibility to infectious disease and atrophy of host cells participating in the defense mechanisms. In the latter case in which there is acute starvation, the host tissues are catabolized for energy production; this results in rapid loss of weight but it hardly affects the balance between the various nutrients "consumed" (which are derived in part from catabolism of the individual's own tissues). In some acute cases, unless there is avitaminosis, the susceptibility to infectious diseases is significantly increased.

3. Air Pollution

For women working in five United States cities Dohan[24] found an association between the incidence of respiratory disease (mostly common colds) lasting more than seven days and the mean concentration of suspended particulate sulphates in the atmosphere.

It is possible that the data of McCarroll, et al.[25,26] are relevant in this respect. These showed for a general population a temporal correlation between concentration of suspended particulate sulphates in the atmosphere and various symptoms of upper respiratory infection.

4 & 5. Emotional and Genetic Effects

These factors probably affect the host resistance; however, because of the limited objectives of this paper, they are assumed to be fortuitously randomized.

23/ Scrimshaw, N. S., C. E. Taylor, and J. E. Gordon. "Interactions of Nutrition and Infection," American Journal of Medical Sciences, Vol. 237, 1959, pp. 367-403.

24/ Dohan, F. C. "Air Pollutants and Incidence of Respiratory Disease," Archives of Environmental Health, Vol. 3, 1961, pp. 387-396.

25/ "Health and the Urban Environment: Health Profiles Versus Environmental..." op. cit.

26/ "Air Pollution and Illness in a Normal Urban Population," op. cit.

Appendix K

Statistical Homogeneity On Disease and Community Factors Affecting Model Parameters

Both Disease and Community exert a statistical effect on the contact rate as well as on the host susceptibility (the "Infective Ratio"). In the Disease and Chronic Conditions Submodel it is assumed that these two effects act independently. This appendix discusses the available evidence pertaining to this assumption.

Appendix K

Pertinence and Validity of the Assumption of Statistical Homogeneity In Both Community and Disease Effects On Each of the Model Parameters

I. INTRODUCTION

If community and disease effects on the model parameters were statistically homogeneous, this homogeneity would permit prediction of unknown values in a matrix of parameter values estimated from the literature.

Any assumption made for the purpose of prediction and explanation should be compatible with current conceptual knowledge in the field of microbiology and realistic in terms of observed biological and behavioral phenomena. Two assumptions considered in this section are: (1) The contact rate is the product of an organism factor, a host factor, and the residual "clean contact rate". (2) The nonspecific susceptibility is the product of an organism factor and a host factor.

II. DEFINITIONS

Any assumption made for the purpose of prediction and explanation should be compatible with current conceptual knowledge in the field of microbiology and realistic in terms of observed biological and behavioral phenomena. Two assumptions considered in this section are: (1) The contact rate is the product of an organism factor, a host factor, and the residual "clean contact rate". (2) The nonspecific susceptibility is the product of an organism factor and a host factor.

B. Definitions

In statistical language, the first assumption means no interaction between the effects of any two of the factors--humidity, temperature change, and crowding--on the contact rate; the second assumption means no interaction between the effects of organism and host on the nonspecific host susceptibility. In the absence of such interaction, the probability of the intersection of events under consideration is the product of the probabilities of the composing events.

The logical procedure is as follows: first, abstract the current microbiological concepts relevant to the organism and host factors in each of the two assumptions; next, confront the two assumptions with the microbiological concepts; and finally, confront the two assumptions with pertinent observations.

Table K-I

PATHOGENIC PROPERTIES OF BACTERIA

Properties of Bacteria	Pathogenic Bacteria	Mechanism
1.0 Surface Components		
1.1[a] Nontoxic, antigenic surface components inhibit phagocytosis; antibodies are protective	Pneumoccus	Capsular polysaccharides.
	Hemophilus influenzae	Capsular polysaccharides.
	Klebsiella pneumoniae	Capsular polysaccharides.
	Group A Streptococcus	M proteins.
	Pasteurella pestis	Capsular antigen.
1.2[a] Nontoxic, antigenic surface components inhibit phagocytosis; antibodies to these components are not protective	Bacillus anthracis	Antibodies are not protective.
1.3[a] Nontoxic, nonantigenic surface components inhibit phagocytosis	Group A and C Streptococcus	Hyaluronic acid capsules.
	Staphylococcus	Secretion of coagulase causes host fibrin to be deposited on the surface of the organism.
1.4[a] Toxic, antigenic surface components (O-antigens) may inhibit phagocytosis	Gram-negative bacteria	The toxic pyrogenic portion of the endotoxin is not antigenic but pyrogenic and potentiates epinephrin action; the nontoxic polysaccharide portion neutralizes the opsonic effect of anti-O-antibody.
2.0 Extracellular Products		
2.1[a] Exotoxic proteins account for the principal pathogenic properties	Corynebacterium diphtheriae	Exotoxin.
2.2[b] Extracellular products are not the principal pathogenic properties but may contribute	Staphylococcus	Leukocidin.
	Proteus	Produces alkaline conditions.
3.0[b] Somatic Endotoxins	Possibly Klebsiella pneumoniae and Neisseria meningitides	
4.0[b] Preference for a Certain Site of the Host May Determine the Degree of Infectivity of an Organism	Organisms in the respiratory tract are more easily disseminated	

Table K-I (Continued)

Properties of Bacteria	Pathogenic Bacteria	Mechanism
5.0[b] Ability to Survive Outside		Resist dryness, light, and unfavorable temperature.

[a] MacLeod, C. M. "Pathogenic Properties of Bacteria and Defense Mechanisms of the Host," Bacterial and Mycotic Infections of Man, 3rd edition, (ed. R. J. Dubos). London: Pitman, 1958, pp. 84-113.

[b] Blair, J. E. "The Staphylococci," Bacterial and Mycotic Infections of Man, 3rd edition, (ed. R. J. Dubos). London: Pitman, 1958, p.315.

[c] These mechanisms may play a role in Klebsiella pneumoniae infection.

Table K-II

MECHANISMS OF HOST DEFENSE

Host Susceptibility	Defense Mechanism
1.0 Entrance Conditions	
1.1[a] Mucus secretion and consequent formation of a mucus blanket are or may be propelled by ciliary movement	---
1.2[b] A viscous mucus may contain inflammatory exudate	Mucus interferes with ciliary movement
1.3[c] Intact epithelium may be disturbed by a concomitant virus infection or noxious gas	Viral infection affects aerial dissemination and susceptibility to bacterial colonization
1.4[a] Cilia of the epithelial cells operate in the respiratory tract	---
1.5[b] The host cells produce lysozym	---
2.0 Inflammatory Reaction	
2.1[b] Fibrin deposit and physiological fibrinolysis	---
2.2[b] Lactic acid concentration	---
2.3[b] Phagocytosis potential	Agranulocytosis interferes with phagocytosis
2.4[b] Local tissue temperature	---
2.5[b] Serum concentration of properdin	---
3.0[b] Clearance by the Reticulo-Endothelial System	---
4.0[b] Specific Immunity by Antibody and Complement Action	---
5.0[b] Hypersensitivity	Koch's phenomenon
6.0[b] Exit Conditions	Cough and sneeze reflex

a/ Bang, F. B. "Mucociliary Function as Protective Mechanism in Upper Respiratory Tract," Bacteriological Reviews, Vol. 25, 1961, pp. 228-236.

b/ MacLeod, C. M. "Pathogenic Properties of Bacteria and Defense Mechanisms of the Host," Bacterial and Mycotic Infections of Man, 3rd edition, (ed. R. J. Dubos). London: Pitman, 1958, pp. 84-113.

c/ Eichenwald, H. F., O. Kotsevalov, and L. A. Fasso. "Some Effects of Viral Infection on Aerial Dissemination of Staphylococci and on Susceptibility to Bacterial Colonization," Bacteriological Reviews, Vol. 25, 1961, pp. 274-281.

Table K-III

BIOLOGICAL MECHANISMS CONCEIVABLY DETERMINING THE CONTACT RATE, THE SUSCEPTIBILITY OF THE POTENTIAL HOST, AND THE CASE-FATALITY RATIO

Description of the Assumption[a]	Reference to Description of the Biological Mechanism[a]
The probability that a susceptible person becomes infected during a certain time period with a given prevalance of infectives:	(f) The "clean contact rate" comprises host behavior[b] and environmental factors[c].
$Pr(A^*\|B^*) = f \cdot g_1$	(g_1) Ability of the organism to survive outside of the host (table K-I: 5.0).
The probability that an infection will lead to the loss of susceptibility in the new host:	(g_2) The quality of the infecting organisms (table K-I 1.1, 1.2, 1.3, 1.4).
$Pr(B^*\|A^*) = g_2 \cdot h_2$	(h_2) Selected host factors that allow for establishment and growth of the organism (table K-II: 1.1, 1.2, 1.3, 1.4, 1.5, 2.1, 2.2, 2.3, 2.4, 2.5).
The probability that an infected person will become infective himself (the propensity of a colonized individual to become an infective):	(g_3) The quality of the infecting organisms (table K-I: 1.1, 1.2, 1.3, 1.4, 4.0).
$Pr(C^*\|B^*) = g_3 \cdot h_3$	(h_3) Selected factors in a new host that allow for establishment, growth, and expulsion of the organisms (table K-II: 2.1, 2.2, 2.3, 2.4, 2.5, 3.0, 4.0, 6.0).[d]
The probability that an infective person will still be infective one time unit later; the number of persons dying during infectivity can be neglected:	(g_4) The quality of the infecting organisms (table K-I: 1.1, 1.2, 1.3, 1.4, 2.1, 2.2).
$Pr(D^*\|C^*) = g_4 \cdot h_4$	(h_4) Selected host factors that allow for continuing expulsion of infective units (table K-II: 2.1 2.2, 2.3, 2.4, 2.5, 3.0, 4.0, 6.0)[e]

[a] For further reference to the symbols f, g_1, etc., see section V, C.

[b] Host behavior includes crowding, coughing and sneezing habits, gregariousness, etc.

[c] Environmental factors include change of temperature, relative humidity indoors, air pollution, etc.

[d] Point 4.0 is of minor importance.

[e] Points 2.0, 2.1, 2.2, 2.3, 2.4 and 2.5 are of minor importance.

Table K-III (Continued)

BIOLOGICAL MECHANISMS CONCEIVABLY DETERMINING THE CONTACT RATE, THE SUSCEPTIBILITY OF THE POTENTIAL HOST, AND THE CASE-FATALITY RATIO

Description of the Assumption	Reference to Description of the Biological Mechanism
The probability that an infective person will die from the disease[f/]	(g_5) The quality of the infecting organisms precipitating the host's death (table K-I: 1.1, 1.2, 1.3, 1.4, 2.1, 2.2, 3.0).
$Pr(E^* \mid C^*) = g_5 \cdot h_5$	(h_5) Selected host factors precipitating death (table K-II: 2.1, 2.2, 2.3, 2.4, 2.5, 3.0, 4.0, 5.0).

[f/] Event (E^*) is the death of an infective person due to the infection.

Before proceeding, however, a notation for the relevant events must be developed. In the process of an infective person infecting susceptibles whereby one or more become infective, five possible events can be listed as follows:

D*' An infective person is the old host.

A* A susceptible who becomes infected is a new host.

B* The new host loses specific susceptibility.

C* The new host becomes infective.

D* The infectivity of the new infective host does not cease within one time unit.

The model parameters can be related to these events as follows:

$$p = Pr(B^*A^* \mid D^{*\prime}) \qquad \alpha D = Pr(D^*C^* \mid B^*) \qquad \text{(K-1)}$$

where "Pr(XY|Z)" is the probability of the intersection of events X and Y, given event Z. Event A* cannot be observed; event B* can be observed only by skin tests or serological tests; and events D*', C* and D* are potentially observable by some convenient index, like a reliable case history (which, however, needs validation by biological methods).

III. LOGICAL PROCEDURE

A. Abstract of Current Microbiological Concepts

Table K-I summarizes most of the pathogenic properties of bacteria relevant to organism and host factors in the two assumptions, and Table K-II summarizes most of the relevant mechanisms of host defense. The examples of pathogenic bacteria, added for clarity to Table K-I, are not limited to the diseases discussed in this paper.

Table K-II lists specific events in the probability chain that affect the contact rate and the nonspecific host susceptibility and, at the same time, refer to biological phenomena.

B. Confrontation of the Two Assumptions with Microbiological Concepts

The two assumptions can be expressed in microbiological terms as follows. The "battle" between host and organism during an infection is represented in five "epochs" given as subscripts 1-5 in Table K-III. In addition, let each epoch of the battle be divided into a large number of very short consecutive stages, 0, 1, 2, 3, ... t ... z, which are added as subscripts to the capital letters below.

Let event A^*_z (table K-III) be conditionally determined by the previous event A^*_{z-1} and by the mutually independent events $G_{1,z-1}$ and F_{z-1} so that

$$\Pr\left(A^*_z \mid A^*_{z-1}\right) = \Pr\left(G_{1,z-1} \mid A^*_{z-1}\right) \Pr\left(F_{z-1} \mid A^*_{z-1}\right) . \qquad \text{(K-2)}$$

Also let

$$\Pr\left(A^*_{z-1} \mid A^*_{z-2}\right) = \Pr\left(G_{1,z-2} \mid A^*_{z-2}\right) \Pr\left(F_{z-2} \mid A^*_{z-2}\right), \text{ etc.} \qquad \text{(K-3)}$$

Thus, a sequence of events can be written in reversed order of occurrence as follows

$$A^*_z,\ A^*_{z-1},\ A^*_{z-2},\ \cdots\ A^*_t,\ \cdots\ A^*_1,\ A^*_o .$$

According to an elementary probability rule,

$$\Pr\left(A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) = \Pr\left(A^*_{t-1} \mid A^*_{t-2}\right) \Pr\left(A^*_t \mid A^*_{t-1}\, A^*_{t-2}\right) . \qquad \text{(K-4)}$$

Substituting equation (K-2) into (K-4),

$$\Pr\left(A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) = \Pr\left(G_{1,t-2} \mid A^*_{t-2}\right) \Pr\left(F_{t-2} \mid A^*_{t-2}\right) \Pr\left(A^*_t \mid A^*_{t-1}\, A^*_{t-2}\right) \qquad \text{(K-5)}$$

In analogy to equation (K-2),

$$\Pr\left(A^*_t \mid A^*_{t-1}\, A^*_{t-2}\right) = \Pr\left(G_{1,t-1} \mid A^*_{t-1}\, A^*_{t-2}\right) \Pr\left(F_{t-1} \mid A^*_{t-1}\, A^*_{t-2}\right) . \qquad \text{(K-6)}$$

Substituting equation (K-6) into (K-5),

$$\begin{aligned}\Pr\left(A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) &= \Pr\left(G_{1,t-2} \mid A^*_{t-2}\right) \Pr\left(F_{t-2} \mid A^*_{t-2}\right) \\ &\quad \Pr\left(G_{1,t-1} \mid A^*_{t-1}\, A^*_{t-2}\right) \Pr\left(F_{t-1} \mid A^*_{t-1}\, A^*_{t-2}\right) \qquad \text{(K-7)} \\ &= \Pr\left(G_{1,t-1}\, G_{1,t-2} \mid A^*_{t-1}\, A^*_{t-2}\right) \Pr\left(F_{t-1}\, F_{t-2} \mid A^*_{t-1}\, A^*_{t-2}\right)\end{aligned}$$

This argument, as given from equation (K-4) through (K-6), can be extended so that

$$\begin{aligned}\Pr\left(A^*_{t+1}\, A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) &= \Pr\left(A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) \Pr\left(A^*_{t+1} \mid A^*_t\, A^*_{t-1}\, A^*_{t-2}\right) \\ &= \Pr\left(A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) \Pr\left(G_{1,t} \mid A^*_t\, A^*_{t-1}\, A^*_{t-2}\right) \\ &\quad \Pr\left(F_t \mid A^*_t\, A^*_{t-1}\, A^*_{t-2}\right) \qquad \text{(K-8)}\end{aligned}$$

Substituting equation (K-7) into K-8),

$$\begin{aligned}\Pr\left(A^*_{t+1}\, A^*_t\, A^*_{t-1} \mid A^*_{t-2}\right) &= \Pr\left(G_{1,t}\, G_{1,t-1}\, G_{1,t-2} \mid A^*_t\, A^*_{t-1}\, A^*_{t-2}\right) \\ &\quad \Pr\left(F_t\, F_{t-1}\, F_{t-2} \mid A^*_t\, A^*_{t-1}\, A^*_{t-2}\right) .\end{aligned}$$

Equation (K-1) $p = \Pr\left(B^*\, A^* \mid D^{*\prime}\right)$ can now be written as

$$\begin{aligned}p = \Pr\Bigg[&\left(G_{2,z-1}\, G_{2,z-2} \cdots G_{2,o} \mid B^*_{z-1}\, B^*_{z-2} \cdots B^*_o\right) \\ &\left(G_{1,z}\, G_{1,z-1} \cdots G_{1,o} \mid A^*_z\, A^*_{z-1} \cdots A^*_1\, D^{*\prime}\right)\Bigg] \cdot \\ \Pr\Bigg[&\left(H_{2,z-1}\, H_{2,z-2} \cdots H_{2,o} \mid B^*_{z-1}\, B^*_{z-2} \cdots B^*_o\right) \\ &\left(F_z\, F_{z-1} \cdots F_o \mid A^*_z\, A^*_{z-1} \cdots A_1\, D^{*\prime}\right)\Bigg] .\end{aligned} \qquad \text{(K-9)}$$

A similar argument applies to the second part of equation (K-1) $\alpha D = Pr(D^* C^* B^*)$.

The equations are applicable to the list of biological mechanisms in Table K-III. Capital letters G_j, H_j, and F designate certain events; the small letters g_j, h_j, and f represent the probability of intersection of all events G_j, H_j and F respectively, when the conditional probabilities of these events are considered. These definitions give the biological meaning to all events discussed.

Equation (K-9) and the parallel equation for the nonspecific human resistance can now be put into words: If, in each of the five probabilities listed in Table K-III, there is conceptual independence between the organism factors, the host factors, and the "clean contact rate" as they influence events, then there is independence among the respective probabilities of intersection of conditionally determined events due to the host, due to the organism and due to the "clean contact rate." Thus, in such a case, the multiplication assumptions of section A, at the beginning of this appendix, would be consistent with current biological knowledge.

In this paper it does not seem feasible to go into detailed description of the accumulated knowledge concerning the interaction between each two of the factors listed in Tables K-I and K-II. A "rough and ready" conclusion would be that there is considerable interaction; but, in view of the multiplicity of factors involved, the assumption that the various interactions will cancel each other out does not seem far from reality.

C. Confrontation of the Two Assumptions with Observations

The assumption of no interaction between disease and community effects can be tested by analysis of variance of observed data with respect to either the contact rate or the nonspecific host resistance.

Data representing an index of the contact rate (as discussed in Chapter 3, Section III, Subsection D) are given in Table K-IV. They apply to measles, whooping cough, and diptheria in 19 large United States cities from 1901 through 1949. Since the data are approximately homoscedastic and normally distributed, the analysis of variance is appropriate. The result is given in Table K-V. No significant interaction could be demonstrated at the 5 percent level of significance, but the statistical power of the test is low. Since most diptheria infections occurred at school age, and most measles and whooping cough at preschool ages, there may be some merit in taking the data for measles and whooping cough only and repeating the analysis (Table K-VI). Again, no significant interaction could be demonstrated.

Data for values of the nonspecific host susceptibility are not available. However, it is possible to produce a similar argument on the probability that an infected individual will die from the disease, by using factors g_5 and h_5 instead of g_4 and h_4 (see Table K-III). Thus, if independence between host and organism effects on cause-specific mortality rates can be demonstrated, such a finding would support the concept of independence between these effects on the nonspecific host susceptibility. In the selection of data the following considerations should be taken into account:

1) Curative or preventive medical treatment should not appreciably influence the disease in its incidence, duration, or fatality.
2) The disease should be readily distinguishable from other diseases and as a cause of death.
3) Reporting should be complete, or at least consistert, when different years are compared.
4) The age groups examined in different communities should be comparable.

Pascua[1/] published rates of reported mortality from measles, whooping cough, diphtheria, and scarlet fever for several European countries between 1900 and 1950. These data can be subjected to a two-way analysis of variance with replications for different year of reporting. Because the raw data are not homoscedastic and their variances are approximately proportional to the square of their means, the data were transformed into their logarithms. The resulting "analysis of variance table" is given in Table K-VII. No significant interaction between disease and country effects could be demonstrated at the 5 percent level of significance.

These tests should be repeated under different conditions. If they invariably would result in "no significant interaction," then the assumption tested could be accepted as a working hypothesis. This working hypothesis is as follows. The observations are consistent with no interaction between the effects of the combined organism factors on the one hand and the combination of host factors, "clean contact rate", and--in the case of mortality rates--facilities for prevention, cure, and reporting on the other hand.

1/ Pascua, M. "Evolution of Mortality in Europe During the Twentieth Century," World Health Organization Epidemiological and Vital Statistics Report, Vol. 4, 1951, pp. 46-137.

Table A-IV

CURVE CROSSINGS PER 25 YEARS[a] BETWEEN LINE OF ANNUALLY REPORTED MORTALITY RATES AND ITS NON-LINEAR REGRESSION LINE

Four Infectious Diseases in 19 Large U. S. Cities for the periods 1901-1925 and 1925-1959[b]

Cities	Observations of Infectious Respiratory Diseases							
	Tuberculosis		Measles		Whooping Cough		Diptheria	
	1st period	2nd period	1901-25	1925-49	1901-25	1925-49	1901-25	1925-49
Baltimore, Md.	2.3	2.3	15	12	16	14	7	9
Boston, Mass.	2.3	3.0	14	12	19	15	9	11
Chicago, Ill.	3.0	1.3	17	21	14	13	11	10
Cleveland, Ohio	---	---	12	16	16	15	10	8
Columbus, Ohio	---	---	11	13	19	16	7	14
Indianapolis, Ind.	4.7	7.3	16	15	15	14	12	8
Kansas City, Mo.	---	---	14	15	16	13	9	8
Los Angeles, Calif.	4.7	10.0	16	18	13	16	10	9
Louisville, Ky.	2.5	2.5	12	15	17	14	10	9
Milwaukee, Wis.	4.0	5.5	10	15	17	14	9	13
Minneapolis, Minn.	---	---	18	15	18	11	11	15
New Orleans, La.	2.5	3.5	14	13	14	14	11	9
New York City, N. Y.	2.7	2.7	16	20	12	11	8	16
Philadelphia, Pa.	---	---	14	19	15	13	3	7
Pittsburgh, Pa.	---	---	18	18	19	17	4	11
Providence, R. I.	2.5	4.5	13	13	13	10	10	14
Saint Louis, Mo.	2.5	5.0	14	15	15	13	12	13
San Francisco, Calif.	3.5	2.0	13	12	14	13	7	10
Washington, D. C.	2.5	4.0	7	16	11	12	10	8
Mean Values	3.05	4.27	13.89	15.32	15.42	13.58	8.95	10.63
Mean Duration of Complete Cycles[c] (in years)	13.7		3.4		3.4		5.1	

[a] The two observation periods for tuberculosis are together up to 150 years

[b] Source: Voors, A. W. A Modified Soper-Reed-Frost Model as a Guide in Programming the Control of Epidemic Respiratory Infection. Chapel Hill, N. C.: University of North Carolina, Doctoral Dissertation, 1965.

[c] $\frac{2 \times 25}{\text{(sum of mean values)}}$

Table K-V

ANALYSIS OF VARIANCE OF ANNUAL MORTALITY RATES DUE TO MEASLES, WHOOPING COUGH AND DIPHTHERIA IN 13 LARGE U. S. CITIES, 1901-1949[a,b/]

Variance Due to	Sum of Squares	Degrees of Freedom	Mean Squares	Variance Ratios	$F_{critical}$ (α=.05)
Disease	270.9[illegible]	2	135.4743	28.25	3.24
City	68.5387	12	5.7116	1.19	2.01
Interaction DxC	174.9998	24	7.2917	1.52	1.80
Error	187.0000	39	4.7949		
Total	701.4872	77			

a/ U. S. Bureau of the Census: Mortality Statistics. Washington, D. C.: U. S. Gov't Printing Office, published annually, 1901-1936.

b/ U. S. Bureau of the Census: Vital Statistics of the U. S. Washington, D. C.: U. S. Gov't Printing Office, published annually, 1937-1949.

Table K-VI

ANALYSIS OF VARIANCE OF ANNUAL MORTALITY RATES DUE TO WHOOPING COUGH AND DIPHTHERIA IN 13 LARGE U. S. CITIES, 1901-1949[a,b/]

Variance Due to	Sum of Squares	Degrees of Freedom	Mean Squares	Variance Ratios	$F_{critical}$ (α=.05)
Disease	1.5922	1	1.5922	0.35	4.23
City	86.3077	12	7.0673	1.58	2.34
Interaction DxC	113.4923	12	9.4577	2.11	2.18
Error	116.5000	26	4.4808		
Total	316.0577	51			

a/ U. S. Bureau of the Census: Mortality Statistics. Washington, D. C.: U. S. Gov't Printing Office, published annually, 1901-1936.

b/ U. S. Bureau of the Census: Vital Statistics of the U. S. Washington, D. C.: U. S. Gov't Printing Office, published annually, 1937-1949.

Table K-VII

ANALYSIS OF VARIANCE OF AGE-SPECIFIC MORTALITY RATES DUE TO WHOOPING COUGH, SCARLET FEVER, DIPHTHERIA AND MEASLES FOR ENGLAND AND WALES, ITALY, NORWAY AND NETHERLANDS AS REPORTED FOR THE YEARS 1900, 1910, 1920, and 1930[a/]

Variance Due to	Sum of Squares	Degrees of Freedom	Mean Squares	Variance Ratios	$F_{critical}$ (α=.05)
Disease	3.553425	3	1.184475	13.70	2.81
Country	3.149213	3	1.049738	12.14	2.81
Interaction DxC	1.144137	9	0.127126	1.47	2.09
Error	4.149000	48	0.086438		
Total	11.995775	63			

a/ Pascua, M. "Evolution of Mortality in Europe During the Twentieth Century," World Health Organization Epidemiological and Vital Statistics Report, Vol. 4, 1951, pp. 36-137.

Appendix K-TAB

Interaction Between the Effects of the Various Host Defense Systems on Infection Incidence in the Case of Chronic Protein Malnutrition

Appendix K-TAB

Interaction Between the Effects of the Various Host Defense Systems on Infection Incidence in the Case of Chronic Protein Malnutrition

Scrimshaw, et al.,[1/] reviewed the experimental evidence for the existence of dietary influences in the etiology of nonspecific host resistance against bacterial disease.

In an extensive literature survey, they found that most references are in agreement concerning the existence of effects of nutrient deficiency on various aspects of bacterial infections and that these effects act synergistically. Dietary deficiencies are associated with reductions in antibody responses, phagocytic activity, nonspecific protective substances (like lysozym), nonspecific destruction of bacterial toxins, and with alterations in intestinal flora, tissue integrity, and endocrine balance of the host.

Scrimshaw, et al., concluded from such experimental data that host resistance against bacterial infection depends on at least four actions by nutritional deficiencies which are mutually interacting in a synergistic manner. These actions are: (1) facilitating initial invasion of the host by the infectious agent; (2) affecting the agent after it is established in the host tissues; (3) favoring secondary infection; and (4) retarding convalescence after infection. Many of the better controlled experiments concentrate on only one of these actions or on part of one. Thus, negative findings in such experiments do not necessarily exclude the influence of malnutrition on the resulting comprehensive state of health.

1/ Scrimshaw, N. S., C. E. Taylor, and J. E. Gordon. "Interactions of Nutrition and Infection," American Journal of Medical Sciences. Vol. 237, 1959, pp. 367-403.

Appendix L

The Effect of Quarantine

This appendix explores the problem of quarantine. That is, if the probability of epidemiological contact between any postattack community member and an outside person is diminished but non-zero, what are the consequences in terms of epidemic risk?

Appendix L

The Effect of Quarantine

I. DERIVATION OF THE ANALOGUE OF THE SOPER-REED-FROST MODEL FOR UNEQUAL CONTACT RATES

The contact rate of a person is the number of contacts per time unit; as such, the rate is a unique personal time series, comparable to pulse rate or respiration rate. However, unlike a pulse or respiration rate, each contact involves two persons and can be classified as either a within-household or a between-household contact rate. Thus, each person in a community has two attributes: the within-household contact rate and the between-household contact rate; the sum of the two rates is the contact rate.

The influence on the model's predictions exerted by the existence of two categories of contacts (within- and between-household) will be investigated by means of an example.

Let the contact rate λ be 5 per time unit and let the rate be equal for every person of the community. Consider a community of 1000 inhabitants ($N = 1000$), being divided into 100 households, each of 10 members ($n = 10$). Further, let there be 10 infectives in the community so that the community prevalence of infectives $i = 0.010$. The question is: How is the community's crude attack rate A affected by a change in relation between the numbers of within- and between-household contacts?

To answer this question, let λ' be the within-household contact rate and λ'' the between-household contact rate, so that $\lambda = \lambda' + \lambda''$. If there is one infective in the household, let p' be the probability that a susceptible becomes infected from within his household within the time unit, and p' the probability that a susceptible becomes infected from outside of the household within the time unit. Likewise, to include the probability of no contact, let $q' = 1 - p'$ and $q'' = 1 - p''$.

According to Abbey[1/],

$$A = S\{1 - (q')^{in}(q'')^{i(N-n)}\} . \qquad (L\text{-}1)$$

Apply equation (3-2), chapter III, to one of the algebraic definitions of e, the base of the natural logarithm:

$$q^{N-1} = e^{-\lambda} .$$

1/ Abbey, H. "An Examination of the Reed-Frost Theory of Epidemics," Human Biology, Vol. 24, 1952, pp. 201-233.

This equation is valid as long as either the community size N is large enough, or p is small enough (p is the probability of at least one contact between any two community members per unit of time; see Appendix A). The latter condition is sufficient according to a mathematical property of e. Hence, within a household of size n, as long as p is small:

$$e^{-\lambda'} = (q')^{n-1} \qquad \text{(L-2)}$$

where it is stipulated that the proband or index person is included in the household.

To express A in terms of S under various assumptions concerning the proportion of contacts that occurs within the household, suppose that all of the 5 contacts per unit of time are within the household and that all households are completely isolated. According to equation (L-2)

$$e^{-\lambda'} = (q')^{n-1} \; ; \; e^{-5} = (q')^{9} \; ; \; q' = 0.5738 \; .$$

$$e^{-\lambda'} = (q'')^{n-1} \; ; \; e^{-0} = (q'')^{999} \; ; \; q'' = 1.000 \; .$$

According to the binomial distribution of infectives among households, there will be 90 households without infectives, 9 households with one infective, and one with two infectives. Thus, in this case, equation (L-1) becomes

$$\frac{A}{S} = 0.90 \left[1 - (q')^{0}(q'')^{10}\right] + 0.09 \left[1 - (q')^{1}(q'')^{9}\right] + 0.01 \left[1 - (q')^{2}(q'')^{8}\right] \; . \qquad \text{(L-3)}$$

By substituting numbers for symbols and completing the computation,

$$\frac{A}{S} = 0.048 \; .$$

The calculations are repeated for the assumption that more of the contacts take place between households. The results are as follows:

Distribution of Contacts		Value for $\frac{A}{S}$
Within Households	Between Households	
$\lambda' = 0$	$\lambda'' = 5$	0.048
$\lambda' = 1$	$\lambda'' = 4$	0.050
$\lambda' = 2$	$\lambda'' = 3$	0.050
$\lambda' = 3$	$\lambda'' = 2$	0.049
$\lambda' = 4$	$\lambda'' = 1$	0.048
$\lambda' = 5$	$\lambda'' = 0$	0.045

Similar calculations were made for (i) = 0.100 and (i) = 0.001; the results are given in a nomogram (Fig. L-1). It can be concluded that, under the given circumstances--which are fairly representative for most infectious diseases in actual communities--a change in the relationship between rates of within-household and between-household contacts has little influence on the rate at which susceptibles become infected.

II. DERIVATION OF THE ANALOGUE OF THE SOPER-REED-FROST MODEL FOR DEPARTURE FROM THE ASSUMPTION OF ISOLATION OF THE COMMUNITY

An isolated community within a country is much like an isolated household within a community (the latter case was examined in the example in Section I of this appendix). In other words, the rate at which susceptibles become infected is rather insensitive to changes in the relationship between rates of within-household and between-household contacts. The same can be applied to communities of size n within a country of size N. Hence, as long as the values for the parameters λ and αD do not differ much between communities, the prediction of $\frac{i}{\alpha D}$ (by the Soper-Reed-Frost model) is insensitive to departure from the assumption of segregation. In such cases, the generalized version of equation (L-3) is applicable.

III. QUARANTINE AFTER NUCLEAR ATTACK

As long as the sheltered community is free from a certain pathogenic organism, the incidence of infectives is near zero of course. As soon as, in case of a less-than-perfect quarantine, such a pathogenic organism would get established, the above developed model shows that an incidence equal to the outside incidence could be expected. However, if the outside population were not exposed to radiation and only the sheltered population were, the incidence in the latter population would exceed that of the outside population due to increased susceptibility.

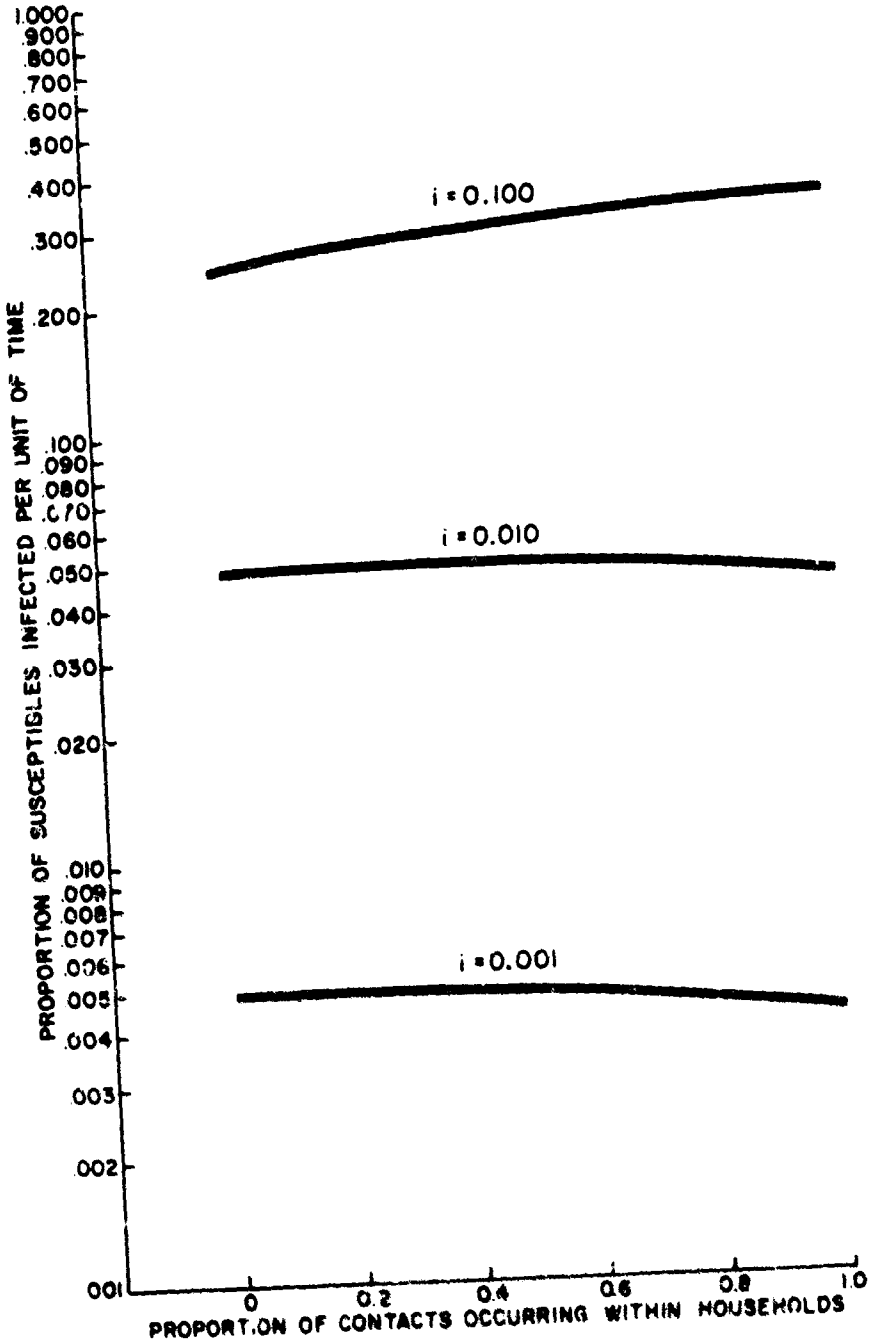


Fig. L-1. Rate of Infection by Proportion of Contacts Occurring Within Households for Various Levels of Prevalence of Infectives (i) Based on a Population of 1,000 Persons Equally Divided Over 100 Households in which Each Person Contacts Five Others Per Unit of Time).

Appendix M

Composition of the Medical Treatment Packages

This appendix contains components of Treatment Packages used as inputs in the Immediate Effects Submodel.

Table 4-1

COMPOSITION OF INJURY TREATMENT PACKAGES
(Column figures indicate number of units of each item)

Treatment Package Items		Injury Packages*															
		Severe Burns	Abdominal	Chest and Abdominal	Pelvic	General Shock	Face and Neck	Arm & Shoulder	Moderate Burns	Femur Fracture	Open Wounds	Head	Hand Fracture	Foot Fracture	Psychiatric	Eye	Vertebrae and Spinal
Number	Name	BI	A	T	P	S	M	AF	BA	LF	W	H	HF	FF	Y	E	V
1	Oxytetracycline, Oral	2	3	3	3		2		2		3						
2	Combined Item A**	1	100	100	50		28		1		10						
3	Dressings, Assortment						2	1	1	1			1	1			
4	Dextrose, Saline 5%	5	10	5	1	6											
5	Elastic Bandages	10						2	1	3		1					
6	Adhesive Tape							1		1	1		1	1			
7	Catheters, Foley	½	2			1											1/10
8	Hypodermic Needles, Assortment		2	1	1	1											
9	Scalpel Blades							1									
10	Phenobarbital Parenteral		2	6								1					
11	Dextrose Water		10		2	6											
12	Sodium Pentobarbital	1										2					

Table M-1 (Continued)

COMPOSITION OF INJURY TREATMENT PACKAGES
(Column figures indicate number of units of each item)

Treatment Package Items		Injury Packages*															
		Severe Burns	Abdominal	Chest and Abdominal	Pelvic	General Shock	Face and Neck	Arm & Shoulder	Moderate Burns	Minor Fracture	Open Wounds	Head	Hand Fracture	Foot Fracture	Psychiatric	Eye	Vertebrae and Spinal
Number	Name	BI	A	T	P	S	M	AF	BA	LF	W	H	HF	FF	Y	E	V
13	Oral Electrolyte Solution	3				2			1								
14	Combined Item B		1	1													
15	Combined Item C						1					1					
16	Tetanus Toxoid						1				10						
17	Arm Slings							1					1				
18	Dextran, 6%	2	1			2											
19	Dressings, Assortment III	1									1						
20	Gantrisin Tablets				2												1
21	Sodium Hydrochloride				1											1	
22	Combined Item D									1				;			
23	Demerol		14						1								
24	Liquid Soap	1							1								

Table M-1 (Continued)

COMPOSITION OF INJURY TREATMENT PACKAGES
(Column figures indicate number of units of each item)

Treatment Package Items		Injury Packages*															
Number	Name	Severe Burns	Abdominal	Chest and Abdominal	Pelvic	General Shock	Face and Neck	Arm & Shoulder	Moderate Burns	Femur Fracture	Open Wounds	Head	Hand Fracture	Foot Fracture	Psychiatric	Eye	Vertebrae and Spinal
		BI	A	T	P	S	M	AF	BA	LF	W	H	HF	FF	Y	E	V
25	Morphine Sulfate	1															
26	Combined Item E		1														
27	Combined Item F				1												
28	Combined Item G					1											
29	Combined Item H						1										
30	Combined Item I							1									
31	Aspirin								1								
32	Combined Item J									1							
33	Combined Item K										1						
34	Combined Item L											1					
35	Hand and Finger Splints												1				
36	Chlorpromazine, Oral														1		
37	Combined Item M															1	

Table M-1 (Continued)

COMPOSITION OF INJURY TREATMENT PACKAGES

Key to Column Headings: BI-Burns of second and third degree covering more than 15 percent of the body; A-Abdominal; T-Thoracic; P-Pelvic; G-Genitourinary; S-Shock; M-Maxillifacial; AF-Fractures of the upper extremities except the hands; BA-Burns of first degree and ambulatory cases of second and third degree covering 15 percent or less of the body; LF-Fracture of the upper leg; W-Open wounds; H-Head; HF-Fractures of the hand; FF-Fractures of the lower leg and/or foot; Y-Psychiatric; E-Eye; V-Vertebrae and spinal cord.

Entries in columns refer to the number of units of an item as defined in Table M-II.

* Units for each item are described in detail in Table M-II.

** The items comprising these combined items (A, B, C, ...) are given in Table M-II.

Table M-II

EXPENDABLE ITEMS IN MEDICAL TREATMENT PACKAGE

(Each Item is Replaceable by Available Substitutes)

Item No.	Unit Nomenclature	Composition of a Treatment Package Unit: Size	Number
1	Oxytetracycline, (film-coated tablets)	250 Mgm.	14
2	Combined Item A		
	Tetracycline Hydrochloride Powder	100 Mgm.	1
	Sterile Water (Ampules)	5 Ml.	5
3	Dressings, Gauze, Assortment I		
	Pad	1 yd.^{2}	12
	Pad	1 yd.^{2}	6
	Compress	1 yd.^{2}	1
	Compress	1 yd.^{2}	1
	Roll Bandage	4" x 10 yds.	1
	Roll Bandage	6" x 5 yds.	1
4	Dextrose, Saline 5%	Liter	1
5	Elastic Bandage, Roll	3" x 25 yds.	1
6	Adhesive Tape, Roll	12" x 10 yds.*	1
7	Catheters, Foley		
	#20	--	1
	#24	--	1
	#16	--	1
8	Hypodermic Needle Assortment		
	25 gauge	1"	10
	20 gauge	1-1/2	5
	18 gauge	2"	5
	15 gauge	2"	2
9	Scalpel Blade	#20	1
10	Sodium Phenobarbital, Parenteral (Ampules)	130 Mgm.	8
11	Dextrose Water 5%	Liter	1
12	Sodium Pentobarbital, Parenteral (Bottles)	100 Mgm.	5
13	Oral Electrolyte Solution (Packet)	4.5 Grams	7 x 130 = 2 lbs in shelter kit

*Each roll cut in following widths: 4 x 1/2"; 3 x 1"; 2 x 2"; 1 x 3"

Table M-II (Continued)

14	Combined Item B		
	Dressings, Gauze, Assortment II	4" x 4"	100
	Pad	4" x 4"	100
	Pad	8" x 10"	100
	Compress	22" x 18"	100
	Roll Bandage	4" x 10 yds.	6
	Roll Bandage	6 x 5 yds.	4
	Syringes (Glass)	2 Ml.	5
	Blood Collecting Tube (Suction)	---	1
	Polyethylene Tubing	---	1
	Size 20	---	1
	Size 18	---	1
	Size 15	---	1
	Procain Hydrochloride 1% (Ampule)	30 Ml.	1
15	Combined Item C		
	Tracheotomy Tube, Silver Plated		
	Size 5	---	1/10
	Size 6	---	1/10
	Size 7	---	1/10
	Oral Airways	---	1/5
16	Tetanus Toxoid Alum Precipitated	5 Ml.	1/10
17	Arm Slings	---	1
18	Dextran 6%	Liter	2
19	Dressing, Gauze, Assortment III		
	Pad	4" x 4"	100
	Pad	8" x 10"	10
	Compress	22" x 18"	10
	Compress	22" x 36"	10
	Roll Bandage	4" x 10 yds.	10
	Roll Bandage	6" x 5 yds.	10
20	Gantrisin Tablets (Tablets)	500 Mgm.	40
21	Sodium Hydrochloride Solution	5 oz.	1
22	Combined Item D		
	Crutches, Wooden	Pair	1
	Padded Wooden Splints	3' x 3" x 1/4"	4
23	Demerol Tablets--Mepiridine Hydrochloride (narcotic)	100 Mgm.	6
24	Liquid Soap (Plastic Bottles)	5 oz.	1
25	Morphine Sulfate (Tablets)	16 Mgm.	10
26	Combined Item E		
	Dextran 6%	Liter	2
	Rubber Drains	1/2"	2
	Catheters, French (Metal)		
	16 Fr.	---	1
	24 Fr.	---	1
	Litmus Paper	---	1
	1/6 Molar Lactate Solution	Liter	1
	Potassium Chloride (Vials)	20 Ml.	20

Table M-II (Continued)

27	Combined Item F		
	Syringes, (Glass)		
	Each	5 Ml.	1
	Each	20 Ml.	1
	Contrast Solution for Cystograms		
	Ampules	30 Ml.	1
28	Combined Item G		
	Serum Albumin	59 Grams	1
	Blood Collection and Dispensing Set*	450 Ml.	1
29	Combined Item H		
	Endotracheal Tubes		
	34 Fr.	---	1/10
	36 Fr.	---	1/10
	40 Fr.	---	1
30	Combined Item I		
	Sheet Wadding, Roll	5"	1
	Padded Wood Splints (Sheets)	2" x 15"	18
31	Aspirin (Tablets)	500 Mgm.	12
32	Combined Item J		
	Thomas Splints		
	Full Ring	---	1/2
	Half Ring	---	1/2
	Elastic Bandage, Roll	6" x 25 yds.	3
	Plaster Splints (Sheets)	5' x 30"	24
	Cotton Wadding, Roll	5"	3
	Padded Wood Splints (Board)	6' x 4" x 1/2"	2
33	Combined Item K		
	Lidocaine Hydrochloride Injection 1% (Ampules)	5 Ml.	25
	Tetanus Antitoxin (Vials)	20,000 Units	1/4
	Epinephrine (Ampules)	1 Ml.	1/10
	Diphenhydramine Hydrochloride (Capsules)	25 Mgm.	1
34	Combined Item L		
	Dressings, Gauze, Assortment IV		
	Roll Bandage	4" x 10 yds.	12
	Pad (8 Ply)	4" x 4"	12
	Caffein Sodium Benzoate Injection (Ampules)	5 Ml.	1
	Dilantin (Capsules)	100 Mgm.	10
	Disposable Razor Blades	---	1
35	Metal Hand and Finger Splint (Aluminum and Foam Rubber)	---	1
36	Chlorpromazine, (Tablets)	25 Mgm.	400

* Includes Anticoagulant

Table M-II (Continued)

37	Combined Item M		
	Procaine 1% Ointment (Tubes)	5 Grams	1
	Lidocaine Ophthalmic Ointment 5% (Tubes)	2 oz.	1
	Tetracycline Ophthalmic Ointment (Tubes)	2 oz.	1
	Eye Patches	---	5
	Cotton Applicator	---	1

Source: Model 62 Civil Defense Emergency Hospital, Component Listing and Storage Data (Washington, D. C.: Public Health Service, 1964), Passive, and Therapeutic Guide for the Civil Defense Emergency Hospital Pharmaceuticals (Washington, D. C.: Public Health Service, 1964).

THE RESEARCH TRIANGLE INSTITUTE, Research Triangle Park, North Carolina
Work Order OCD-PS-64-227 - Final Report R-OU-332
OCD Work Unit 3432A - Public Health Service Contract No. PH-110-67

National Emergency Health Preparedness Study Including the Development and Testing of a Total Emergency Health Care System Model
Project Personnel: E.L. Hill, A.W. Voors, R.O. Lyday, J.N. Pyecha, J.B. Hallan, J.T. Ryan and C. N. Dillard
November 1968 (UNCLASSIFIED) 221 pages

This study developed a Total Emergency Health Care System Model that can be used to study postattack problems in medical preparedness planning for a single locality. The model consists of two submodels that analyze medical system effectiveness, measured by survivors, as a function of medical resources (personnel, facilities and supplies) and their employment. The Immediate Effects Submodel analyzes the first 60 days after attack and is applicable to those casualties that survive the initial weapon effects. Casualty types resulting from a specified attack and available medical resources are input. A prognosis based on injury type, availability of appropriate medical personnel, and available medical supplies is applied to these casualties. The number of deaths and survivors, along with the utilization of medical supplies and personnel, are output. The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats to survivors of the 60 days postattack period throughout the ensuing year. Using a mathematical model of infection, survivors are subjected to the risks of becoming infected by one or more of 16 communicable diseases. A prognosis function is then applied to the infectives. The model output specifies the number of fatalities and the consumption of medical resources by five-day periods for each disease. New Orleans, Louisiana was used as a case study. The hypothetical attack was a surface burst by a 1.5 MT weapon approximately 9 miles south of the center of the city. The results of the case study indicate that relatively unlimited resources prevent few deaths among direct effect injured. However, large numbers of epidemic deaths are preventable in the late postattack environment. Since these preventable deaths are highly dependent upon medical resource availability, the importance of preattack medical resource planning and stockpiling of supplies is indicated.

THE RESEARCH TRIANGLE INSTITUTE, Research Triangle Park, North Carolina
Work Order OCD-PS-64-227 - Final Report R-OU-332
OCD Work Unit 3432A - Public Health Service Contract No. PH-110-67

National Emergency Health Preparedness Study Including the Development and Testing of a Total Emergency Health Care System Model
Project Personnel: E.L. Hill, A.W. Voors, R.O. Lyday, J.N. Pyecha, J.B. Hallan, J.T. Ryan and C. N. Dillard
November 1968 (UNCLASSIFIED) 221 pages

This study developed a Total Emergency Health Care System Model that can be used to study postattack problems in medical preparedness planning for a single locality. The model consists of two submodels that analyze medical system effectiveness, measured by survivors, as a function of medical resources (personnel, facilities and supplies) and their employment. The Immediate Effects Submodel analyzes the first 60 days after attack and is applicable to those casualties that survive the initial weapon effects. Casualty types resulting from a specified attack and available medical resources are input. A prognosis based on injury type, availability of appropriate medical personnel, and available medical supplies is applied to these casualties. The number of deaths and survivors, along with the utilization of medical supplies and personnel, are output. The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats to survivors of the 60 days postattack period throughout the ensuing year. Using a mathematical model of infection, survivors are subjected to the risks of becoming infected by one or more of 16 communicable diseases. A prognosis function is then applied to the infectives. The model output specifies the number of fatalities and the consumption of medical resources by five-day periods for each disease. New Orleans, Louisiana was used as a case study. The hypothetical attack was a surface burst by a 1.5 MT weapon approximately 9 miles south of the center of the city. The results of the case study indicate that relatively unlimited resources prevent few deaths among direct effect injured. However, large numbers of epidemic deaths are preventable in the late postattack environment. Since these preventable deaths are highly dependent upon medical resource availability, the importance of preattack medical resource planning and stockpiling of supplies is indicated.

THE RESEARCH TRIANGLE INSTITUTE, Research Triangle Park, North Carolina
Work Order OCD-PS-64-227 - Final Report R-OU-332
OCD Work Unit 3432A - Public Health Service Contract No. PH-110-67

National Emergency Health Preparedness Study Including the Development and Testing of a Total Emergency Health Care System Model
Project Personnel: E.L. Hill, A.W. Voors, R.O. Lyday, J.N. Pyecha, J.B. Hallan, J.T. Ryan and C. N. Dillard
November 1968 (UNCLASSIFIED) 221 pages

This study developed a Total Emergency Health Care System Model that can be used to study postattack problems in medical preparedness planning for a single locality. The model consists of two submodels that analyze medical system effectiveness, measured by survivors, as a function of medical resources (personnel, facilities and supplies) and their employment. The Immediate Effects Submodel analyzes the first 60 days after attack and is applicable to those casualties that survive the initial weapon effects. Casualty types resulting from a specified attack and available medical resources are input. A prognosis based on injury type, availability of appropriate medical personnel, and available medical supplies is applied to these casualties. The number of deaths and survivors, along with the utilization of medical supplies and personnel, are output. The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats to survivors of the 60 days postattack period throughout the ensuing year. Using a mathematical model of infection, survivors are subjected to the risks of becoming infected by one or more of 16 communicable diseases. A prognosis function is then applied to the infectives. The model output specifies the number of fatalities and the consumption of medical resources by five-day periods for each disease. New Orleans, Louisiana was used as a case study. The hypothetical attack was a surface burst by a 1.5 MT weapon approximately 9 miles south of the center of the city. The results of the case study indicate that relatively unlimited resources prevent few deaths among direct effect injured. However, large numbers of epidemic deaths are preventable in the late postattack environment. Since these preventable deaths are highly dependent upon medical resource availability, the importance of preattack medical resource planning and stockpiling of supplies is indicated.

THE RESEARCH TRIANGLE INSTITUTE, Research Triangle Park, North Carolina
Work Order OCD-PS-64-227 - Final Report R-OU-332
OCD Work Unit 3432A - Public Health Service Contract No. PH-110-67

National Emergency Health Preparedness Study Including the Development and Testing of a Total Emergency Health Care System Model
Project Personnel: E.L. Hill, A.W. Voors, R.O. Lyday, J.N. Pyecha, J.B. Hallan, J.T. Ryan and C. N. Dillard
November 1968 (UNCLASSIFIED) 221 pages

This study developed a Total Emergency Health Care System Model that can be used to study postattack problems in medical preparedness planning for a single locality. The model consists of two submodels that analyze medical system effectiveness, measured by survivors, as a function of medical resources (personnel, facilities and supplies) and their employment. The Immediate Effects Submodel analyzes the first 60 days after attack and is applicable to those casualties that survive the initial weapon effects. Casualty types resulting from a specified attack and available medical resources are input. A prognosis based on injury type, availability of appropriate medical personnel, and available medical supplies is applied to these casualties. The number of deaths and survivors, along with the utilization of medical supplies and personnel, are output. The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats to survivors of the 60 days postattack period throughout the ensuing year. Using a mathematical model of infection, survivors are subjected to the risks of becoming infected by one or more of 16 communicable diseases. A prognosis function is then applied to the infectives. The model output specifies the number of fatalities and the consumption of medical resources by five-day periods for each disease. New Orleans, Louisiana was used as a case study. The hypothetical attack was a surface burst by a 1.5 MT weapon approximately 9 miles south of the center of the city. The results of the case study indicate that relatively unlimited resources prevent few deaths among direct effect injured. However, large numbers of epidemic deaths are preventable in the late postattack environment. Since these preventable deaths are highly dependent upon medical resource availability, the importance of preattack medical resource planning and stockpiling of supplies is indicated.

Unclassified
Security Classification

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author)	2a. REPORT SECURITY CLASSIFICATION
Research Triangle Institute P.O. Box 12194 Research Triangle Park, North Carolina 27709	Unclassified 2b. GROUP

3. REPORT TITLE

NATIONAL EMERGENCY HEALTH PREPAREDNESS STUDY INCLUDING THE DEVELOPMENT AND TESTING OF A TOTAL EMERGENCY HEALTH CARE SYSTEM MODEL

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)

Final Report: May 1967 - August 1968

5. AUTHOR(S) (First name, middle initial, last name)

Project Personnel: Edward L. Hill, Antonie W. Voors, Russell O. Lyday, Jr., John N. Pyecha, Jerome B. Hallan, Joseph T. Ryan and Clarence N. Dillard

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
November 1968	221	105

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
Public Health Service Contract No. PH-110-67 b. PROJECT NO. Work Order OCD-PS-64-227 c. OCD Work Unit 3432A d. RTI Project OU-332	R-OU-332 9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)

10. DISTRIBUTION STATEMENT

This document has been approved for public release and sale; its distribution is unlimited.

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	Office of Civil Defense Office of the Secretary of the Army Washington, D. C. 20310

13. ABSTRACT

This study developed a Total Emergency Health Care System Model that can be used to study postattack problems in medical preparedness planning for a single locality. The model consists of two submodels that analyze medical system effectiveness, measured by survivors, as a function of medical resources (personnel, facilities and supplies) and their employment. The Immediate Effects Submodel analyzes the first 60 days after attack and is applicable to those casualties that survive the initial weapon effects. Casualty types resulting from a specified attack and available medical resources are input. A prognosis based on injury type, availability of appropriate medical personnel, and available medical supplies is applied to these casualties. The number of deaths and survivors, along with the utilization of medical supplies and personnel, are output. The Disease and Chronic Conditions Submodel is designed to model the generation and effects of likely disease threats to survivors of the 60 days postattack period throughout the ensuing year. Using a mathematical model of infection, survivors are subjected to the risks of becoming infected by one or more of 16 communicable diseases. A prognosis function is then applied to the infectives. The model output specifies the number of fatalities and the consumption of medical resources by five-day periods for each disease. New Orleans, Louisiana was used as a case study. The hypothetical attack was a surface burst by a 1.5 MT weapon approximately 9 miles south of the center of the city. The results of the case study indicate that relatively unlimited resources prevent few deaths among direct effect injured. However, large numbers of epidemic deaths are preventable in the late postattack environment. Since these preventable deaths are highly dependent upon medical resource availability, the importance of preattack medical resource planning and stockpiling of supplies is indicated.

DD FORM 1 NOV 68 1473 REPLACES DD FORM 1473, 1 JAN 64, WHICH IS OBSOLETE FOR ARMY USE.

Unclassified
Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Casualties						
Diseases						
Medical Supplies						
Medical Personnel						
Radiation Injuries						
Survival						
Wounds and Injuries						
Damage Assessment						
Decision Making						
Effectiveness						
Mathematical Prediction						
Models						
Sensitivity						
Simulation						
Postattack Operations						